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INDEX ISSUE

VOLUME LXVII DECEMBER, 1957 NUMBER 12

JAN 17 1958

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VOL. LXVII DECEMBER, 1957 No. 12

PERILABYRINTHITIS.*

TERENCE CAWTHORNE, F.R.C.S.,

London, England.

During the past 12 years I have seen a number of patients suffering from a severe disturbance of equilibrium, all of whom at some time previously had been subjected to one or more mastoid operations. There was usually a remnant of hearing in the affected ear, and always evidence of vestibular function in the shape of an active fistula sign and a brisk response to caloric stimulation.

The disturbance of balance was often so severe and the gait so bizarre as to arouse suspicion that the condition was functional rather than organic; nevertheless we felt that the underlying cause might well be irregular and unaccustomed fluctuations in vestibular activity as the result of a fistula in the bony labyrinth, usually the lateral canal. Why such a fistula should be followed by the events I have just described in some patients but not in others we do not know; but it may have something to do with the mobility of the stapes. If the stapes is freely mobile then a fistula in the lateral canal will readily permit a wave of movement in the perilymph between it and the oval window, and such movement is likely to stimulate part of the vestibular mechanism. Thus movements of the head, transmitted vibration when walking on a hard pavement, direct pressure on the fenestra, or acoustic pressure from loud sounds may result in a vestibular stimulus.

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*Read at the meeting of the Sixth International Congress of Otolaryngology, Washington, D. C., May 5, 1957.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Oct. 1, 1957.

The first case I recognized was that of a middle aged accountant who came into the clinic holding on to a relative with one arm and supporting himself with a stick on the other side. He staggered slowly towards me and sank thankfully into the examination chair. Once seated he was at ease and cheerful. As soon as he got up again and attempted to walk he was insecure, and had to hold on to anything or anyone to maintain his balance. He had been like this ever since a mastoid operation three years previously. Examination revealed a clean radical mastoid cavity on the left side with a very brisk, one might almost say startling, fistula sign. There was very little hearing in that ear, but the opposite ear was normal. He had been under medical and even psychiatric treatment some some time but had made no progress, and had been unable to do any kind of work since the mastoid operation.

I operated on his left ear, found a fistula in the lateral canal and opened the labyrinth. He was extremely giddy for a few days after the operation, but with appropriate head and balancing exercises he made steady progress, was able to walk steadily and unaided within three weeks, and was back at work within two months.

Another patient, a lady of middle age, had been bedridden for five years because of disturbed balance. She, too, was quite cheerful and gave a history of multiple operations on both ears. On the left side she had a moist radical mastoid cavity with practically no hearing but a very active fistula sign. The right side showed a dry modified radical mastoid cavity with moderate hearing. Exploration of the left ear was advised, and at operation a large fistula was found in the lateral semicircular canal. The labyrinth was destroyed, and within three weeks she was walking about unaided. When she came up to see me a month later I was told that she had resumed her full household duties.

A third patient, a young housewife, had a similar disability but from a different cause. Following a fenestration operation for the relief of deafness due to otosclerosis, the giddiness which usually follows this operation, instead of subsiding as she got up and about, became much worse and was particularly noticeable when she went out shopping in the busy main street of the town in which she lived. She soon found that the noise of traffic became unbearable so that she was unable to go out; she also found that she could no longer endure loud sounds in the home, such as the radio and the rattle of crockery. She became very nervous and apprehensive and dared not venture out unaccompanied. Examination revealed an extremely active fistula sign, and on testing her with a pure tone audiometer she nearly fell off the chair when presented with a 2,000 cycle tone at 80 db., but remained unaffected by other frequencies even when loud.

I concluded that she was suffering from the effects of a Tullio phenomenon. Here again destruction of the labyrinth resulted in a complete cure; though of course the hearing in that ear was lost.

A fourth patient, a man aged 44, was unable to follow his work as a window cleaner because of continuous giddiness after a modified radical mastoid operation. I first saw him after he had been away from work for eighteen months and had attended no less than five different hospitals. I found a fistula in the lateral canal at operation and he, too, returned to his work as a window cleaner within three months of the operation.

These four examples have been selected from 15 patients, all of whom had a disabling disturbance of equilibrium and a fistula into the bony labyrinth. In some the fistula had prob-

ably been present before a mastoid operation and in a few it may have been the result of operation. In all, however, the result was serious for they were unable to follow their occupations, and most of them had been labelled as hysterical.

There is no doubt that by the time I saw them they all exhibited some of the stigmata of a functional nervous disorder, but in all but three, destruction of the offending labyrinth restored their balance and enabled them to return to work.

I have occasionally seen somewhat similar symptoms in connection with Meniere's disease, after intoxication of the vestibular system with streptomycin, and after concussive head injuries with utricular damage; but today we are concerned with those patients who exhibit a gross disturbance of equilibrium following some form of operation on the ear.

Of course these events do not inevitably follow the making of an opening into the vestibular labyrinth. If they did then every patient who is submitted to a fenestration operation would suffer; but, as we all know, this very rarely happens after fenestration, and I believe that the reason is that the stapes is not free to move (Cawthorne, 1949, 1956). If, however, the stapes is unimpeded then this curious syndrome is liable to appear; and it will be further accentuated by an excitable temperament and also by inappropriate management.

We have recognized this disorder for some years now, and Hallpike and I have been in the habit of referring to it as perilabyrinthitis, because it seems to be associated with or to follow infection around the labyrinth.

I now think that it is the result of irregular vestibular activity brought about by a fistula in the bony labyrinth, which allows frequent and unaccustomed fluctuations in vestibular activity and may even result in a vestibular response to an acoustic stimulus (Cawthorne, 1957). Man is not normally accustomed to receiving such impressions from his vestibular sense organ and to having them forced upon his consciousness; and if he is the type of person who is easily upset it may have a profoundly disturbing effect upon him.

The number of patients whom I have seen with this disorder is not large, but the effect of destroying the disordered sense organ is often so dramatic and so effective that I think that it deserves recording.

A full explanation of the underlying cause of the disordered equilibrium is the first step towards recovery. If the condition warrants it, destruction of the offending end organ may be needed, and in all cases a course of head and balancing exercises plays an important part in restoring balance. These exercises were first introduced by Cooksey and myself in 1946 as part of the rehabilitation of patients suffering from post-concussional vertigo and from vertigo following operations on the labyrinth. These exercises are designed to encourage the patient to move his eyes, head and body freely in all directions. Starting gradually the exercises progress daily so that the patient can with confidence execute movements that formerly caused disturbing giddiness. Daily exercises for three weeks, preferably in a class, are usually sufficient. In the later stages it has been found useful to give training in walking, up and down steps, over uneven ground, and with the eyes shut.

A film of this work illustrating the curious abnormalities of gait adopted by these patients, the beneficial effects which follow labyrinthectomy and a course of head exercises, was shown at the Congress under the title, "The Effect Upon Balance and Gait of Disorders of the Vestibular Labyrinth."

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UNILATERAL DEAFNESS.*

ALFRED T. LIEBERMAN, M.D.,†

Baltimore, Md.

INTRODUCTION.

The diagnosis of unilateral deafness is readily made. Often the cause of the impairment is obvious from the physical examination, or can readily be deduced from the patient's history; but when the history and the physical examination fail to yield an explanation of the deafness it usually is difficult to determine the cause of the hearing loss.

In nine years of practice the writer has had 57 patients with unilateral deafness of obscure etiology; they are the basis for the present study. Excluded from the group are the cases of unilateral deafness caused by impacted cerumen, exostosis of the canal wall, congenital or acquired atresia of the external canal, otitis media or sequelae of otitis media; also those cases in which a history of mumps is associated with the onset of the hearing loss, or in which a history of direct trauma or of acoustic trauma is associated with the onset of impaired function. Further excluded from this study are those cases in which the general examination indicates the presence of either a brain lesion or an acoustic nerve tumor as the possible cause of the unilateral hearing loss.

The present study is an attempt to see whether, by analysis of histories and observations of these 57 patients, information can be derived that may in the future aid in determination of the etiology of unilateral deafness.

REVIEW OF LITERATURE.

Review of the literature pertinent to unilateral deafness reveals a variety of etiologic factors reported as responsible for producing unilateral hearing impairment. That mumps

*Submitted as Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1957.

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Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Feb. 20, 1957.

(epidemic parotitis) can cause deafness, either bilateral or unilateral, has long been known. A typical example was reported by Wallerstein[1] in 1928. The patient was an 18-year-old girl who had bilateral parotid involvement. On the fourth day of her illness she complained of intense roaring and deafness in the left ear. There was no pain or vertigo. Examination later showed normal reaction to both caloric and rotation tests, normal hearing on the right and total deafness on the left.

Fowler, E. P., Jr., and Basek, M.,[2] reported 15 cases of total or nearly total deafness in one ear with normal hearing in the other, in a study of 270 children with permanent deafness, all under the age of ten. In eight it followed mumps; in three measles; in two, skull fractures, and in two the causes were unknown.

Silverman, N. M.,[3] described unilateral deafness as a sequel to non-fatal lightning trauma. A 65-year-old woman was struck by lightning and sustained burn injuries to her body and left side of the face. Bleeding from the left ear canal was noted, and subsequent examinations revealed total deafness in the left ear.

West, G. B.,[4] reported a patient who developed unilateral hearing loss following lightning trauma. No appreciable visible trauma to the ear drum was observed, and hearing tests nine months later showed a return to normal hearing levels in the affected ear.

Hughson, W.,[5] in an analysis of 1400 cases of hearing loss of all degrees of impairment, reported 128 cases of unilateral hearing loss, or nine per cent of the total. The cases were classified according to their etiology into the following groups:

1. Infection with complications.

2. Toxic involvement of the neural mechanism of hearing resulting from acute streptococcus infection of the nasopharynx.

3. Trauma, direct and acoustic.

4. Meniere's disease.

5. Acoustic nerve and brain tumors.

6. Virus disease. Mumps, both the parotitic and non-parotitic forms.

Emphasis is placed on the role of mumps in producing unilateral deafness and Enders' studies[6] are cited. The latter author, on the basis of skin sensitivity and complement fixation tests, concluded that mumps may have occurred in 50 per cent of individuals who disclaim any knowledge of the infection.

Rasmussen, H.,[7] in 1949 reported a series of 18 cases of sudden deafness, having no demonstrable cause, occurring in otherwise normal individuals. The disease was in all cases unilateral. He points out that the occurrence of this type of deafness is relatively rare, in that no more than 18 cases were observed over a period of 15 years. In three patients, the acoustic apparatus alone was affected, while in 15 the vestibular was also involved. In discussing the etiology, three cases were thought due to thrombosis of the internal auditory artery or one of its branches, or hemorrhage in the labyrinth due to rupture of a vessel. In one case the possibility of vascular spasm is considered. In the remaining 14 cases the cause is supposed to be one of neuritis of the VIIIth cranial nerve.

In 1950, 16 cases of unilateral inner ear deafness of sudden onset, with and without vestibular symptoms, were reported by Lindsay and Zudema.[8] These authors observed that four cases represented hearing impairments in which the ear complication was associated with a systemic disease. Twelve cases of sudden unilateral inner ear deafness with varying degrees of vestibular disturbance were of uncertain etiology, but were thought most likely to be due to a toxic neuritis.

Williams, H. L., et al.,[9] in 1950 explained the occurrence of inner ear deafness involving low tones rather than high on the basis of an endolymphatic hydrops without vertigo. They postulate the formation of an extra-cellular edema involving primarily the scala media of the cochlea, and believe that the changes are initiated by a form of physical allergy. In their study of 1394 patients having perceptive deafness, 32

met the criteria established for diagnosis of endolymphatic hydrops without vertigo. Of the 32 cases, eight had a unilateral involvement, 24 a bilateral hearing loss.

Fowler, E. P.,[10] in 1950 reported on 26 cases of sudden deafness in which binaural profound or total deafness occurred in six and monaural total deafness in 20. Five of the 26 patients who were examined for "sludging" of the blood showed it distinctly. It was felt that the neural irritation and degenerations causing the sudden deafness, tinnitus and vertigo under discussion were accompanied by and at least in part caused by the sludging phenomena.

Love, J. G.,[11] in 1950 reported a case of unilateral deafness and progressive facial palsy due to an intrapetrous neurofibroma. This observation is of particular interest because of the existence of right unilateral deafness of eight years' duration, and with progressive right facial palsy of two-and-a-half years' duration.

In a study of 1150 cases of otosclerosis in a consecutive series of 2385 patients in whom the primary complaint was deafness, Cawthorne, T.,[12] found that 1123 patients exhibited bilateral deafness and 27 unilateral. This would place the incidence of unilateral clinical otosclerosis at 2.4 per cent of all patients exhibiting clinical otosclerosis in this series, and at slightly over 1 per cent of patients having all types of hearing losses.

In 1952, Hoople, G. D.,[13] reported his observations on 312 otosclerotics and found that unilateral clinical otosclerosis occurred in 38, a percentage of 12.2 per cent. The diagnosis in each instance was made upon the classical signs for the diagnosis of otosclerosis when there is bilateral involvement.

Sacher, H.,[14] reported five cases of sudden unilateral deafness. In two cases the onset of the deafness was preceded by physical effort; one case was a diabetic; one a pregnant woman, and in one case with hypertension, deafness followed hair drying by electric heat. Vestibular examinations showed normal responses in all cases. Full restitution of hearing followed in all cases except one. The author concludes that

blood extravasation in the cochlea was the cause of the sudden deafness in all cases.

Nielsen and Berryman[15] described two patients exhibiting unilateral deafness associated with pontine lesions of vascular origin. In both patients, extensive neurologic changes besides the hearing loss were present.

PRESENTATION OF MATERIAL.

The material comprising the present study is made up of the case histories and audiometric studies of 57 patients with unilateral deafness who were observed during the past nine years. For inclusion in the study group, the following criteria were used:

a. Presence of normal hearing in one ear and hearing loss ranging from moderate to total deafness in the other ear.

b. Normal findings in both ears on otoscopic examination.

c. Absence of otitis media or sequelae of otitis media.

d. Absence of significant abnormalities of the upper air passages and nasopharynx.

e. Exclusion of acoustic trauma, intracranial neoplasm and head injuries as possible causative factors in the unilateral hearing loss.

INCIDENCE.

The 57 patients exhibiting a unilateral hearing loss that met the above criteria were encountered in a total series of 1475 patients on whom audiometric studies were performed because of hearing complaints of various types and degress, a percentage of 3.8.

The difference betwen this incidence and those reported by previous authors may well be due to the criteria used for inclusion of cases.

AGE, SEX AND EAR DISTRIBUTION IN 57 CASES OF UNILATERAL HEARING LOSS.

In the total series of 57 patients with unilateral hearing loss, the youngest patient was eight years old and the oldest 62. There were 26 males and 31 females. The right ear was involved in 23 cases and the left in 34 cases (See Table I).

TYPES OF DEAFNESS.

Analysis of the 57 cases exhibiting unilateral hearing loss showed that a conduction type deafness was present in 34, and a nerve type deafness in 23. The diagnosis of the type deafness present was based on generally accepted otologic principles employing air and bone conduction audiometry and tuning fork tests. Masking of the normal hearing ear was used routinely when testing the affected ear.

TABLE I.

Age, sex and ear distribution in 57 cases of unilateral hearing loss.

No. of Cases	0-9 Yrs.	10-19	20-29	30-39	40-49	50-59	60 and over	Male	Female	Rt. Ear	Lt. Ear
(Total 57)	2	5	9	21	14	5	1	26	31	23	34

UNILATERAL CONDUCTION DEAFNESS.

Examination of the 34 cases of unilateral conduction type deafness showed a rather striking homogeneity. No patient in this group was below the age of 23; one patient was 62, one 58 and one 54 years old; the remaining 31 were between the age of 23 and 50 years. Fifteen patients were males and 19 females. The right ear was involved in 15 cases and the left ear in 19 cases (see Table II).

Duration of Hearing Loss:

The duration of the hearing loss in this group of patients varied from a period of three months prior to being examined, to 15 years. The average duration of the hearing loss was approximately four-and-a-half years.

Tinnitus:

Tinitus of varying types, degrees and constancy was com-

plained of by 20 of the 34 patients showing unilateral conduction hearing loss.

Vertigo:

Dizziness was noted in only three of the patients in this group. In all three the dizziness was relatively minor and was described as momentary episodes of imbalance. No severe dizziness with nausea and vomiting occurred in patients in this group.

Audiometric Pattern:

Another manifestation of the homogeneity of symptoms in patients with unilateral conduction hearing loss was shown in the audiometric pattern. Of the 34 cases in this series, the audiograms of 30 showed findings characteristic and typical of conductive hearing loss (see Fig. 1).

TABLE II.

Age, sex and ear distribution in 34 cases of unilateral conduction type deafness.

No. of Cases	0-9 Yrs.	10-19	20-29	30-39	40-49	50-59	60 and over	Male	Female	Rt. Ear	Lt. Ear
(Total 34)	0	0	7	16	8	2	1	15	19	15	19

The remaining four cases showed evidence of nerve damage superimposed on an underlying pattern of conduction type hearing impairment. This was manifested by high tone hearing loss by air conduction audiometry, and by a lowering of the threshold for bone conduction audiometry below the level expected for a pure conduction hearing loss.

Family History of Deafness:

Thirteen patients gave a positive history of deafness in the immediate family. Sixteen patients denied knowledge of deafness in the family, and in the remaining five patients nothing was known regarding absence or presence of familial deafness.

Two patients in this group with unilateral conduction loss were father (62 years of age) and son (31 years of age). The left ear in each was involved. The hearing loss in the

father had been present for approximately 15 years, and in the son for approximately one year. A striking similarity in their audiograms is present (see Figs. 2, 3).

Another patient in this series, a 29-year-old white male, stated that his father had unilateral hearing loss on the same side as that of the patient. No hearing studies were obtained on the father.

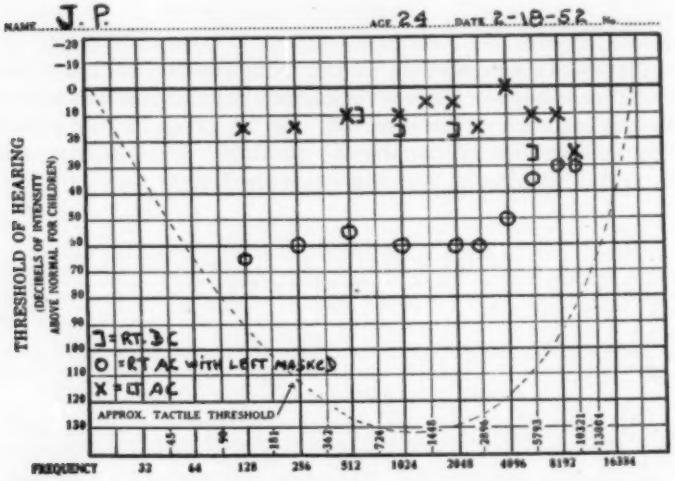

Fig. 1. Audiogram showing unilateral conduction type hearing loss.

ETIOLOGY OF UNILATERAL CONDUCTION HEARING LOSS.

The occurrence of unilateral otosclerosis was reported by Guild, S. R.,[16] in 1944 in a study on histologic otosclerosis. In a series of 1161 patients with serially sectioned temporal bones, 49 had histologic otosclerosis.

Sections of both ears were available for 46 of the 49 otosclerotic patients; the disease was bilateral in 32 and unilateral in 14. Stapedial ankylosis had occurred in only 10 of the 81 otosclerotic ears. In 45 ears an otosclerotic area was less than 0.1 mm. from the stapedial footplate, but an-

kylosis had not occurred. Guild concluded "otosclerosis is often asymptomatic. The disease does not cause impaired hearing unless, or until, ankylosis of the stapedio-vestibular articulation occurs and prevents normal movements of the footplate of the stapes. He should keep in mind the fact that persons with normal hearing may have otosclerotic areas, that the distance of many such areas from the stapes

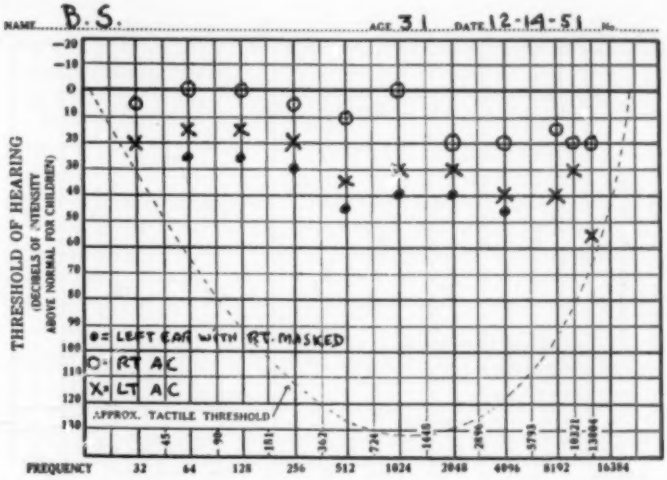

Fig. 2. Audiogram showing unilateral conduction hearing loss in son, age 31.

is less than 0.1 mm., and that growth of the area across the narrow gap would cause deafness."

In the 34 cases exhibiting unilateral conduction hearing loss, relatively little difficulty was encountered in arriving at a diagnosis. In 30 of the 34 cases, the diagnosis was uncomplicated; in four cases, nerve damage was apparently superimposed on the basic otoscerotic process. While it is recognized that a definitive diagnosis can be made only by histologic study of the temporal bones involved, it would seem that in the above 34 patients the most likely basis for the deafness is unilateral otosclerosis.

UNILATERAL NERVE TYPE DEAFNESS.

In the total series of 57 patients with unilateral hearing impairment, 23 patients exhibited impairment of a nerve type. In sharp contrast to the uniformity of symptoms and findings in the patients with conduction type deafness, the patients showing a nerve-type deafness had variable symptoms, findings and etiologic factors. The age distribution

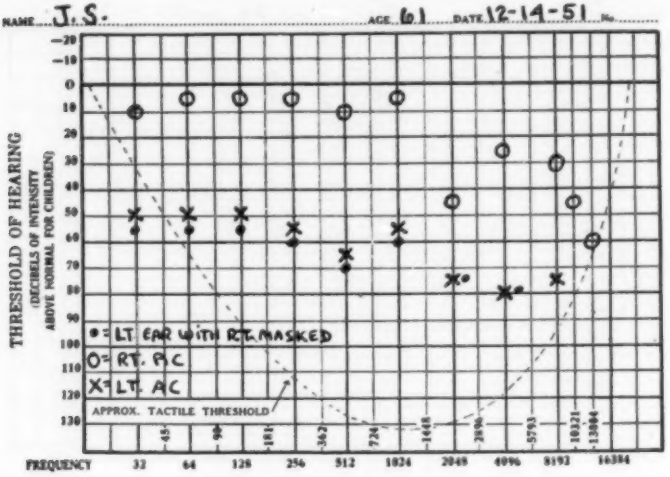

Fig. 3. Audiogram showing unilateral conduction hearing loss in father, age 61.

encompassed all decades of life; the right ear was involved in eight cases and the left in 15. There were 11 males in the series and 12 females (see Table III).

Duration of Hearing Loss:

Duration of the hearing impairment varied considerably. One patient was seen two hours after the onset of his hearing loss; three patients were seen at five, eight and ten days respectively after hearing impairment became noticeable. Eight patients were seen whose hearing loss began within a period of three months to one year previously. In four

patients, the hearing loss had been noted for a period of one to five years, and in seven patients the hearing loss had existed from six to 25 years.

Categories of Patients with Unilateral Nerve Deafness:

Analysis of the case histories and audiometric studies of the 23 patients exhibiting unilateral nerve deafness revealed that five different categories or syndromes could be recognized (see Table IV).

GROUP I: *Sudden, acute onset of deafness without apparent precipitating cause.*

Seven patients were encountered in whom the onset of the

TABLE III.

Age, sex and ear distribution in 23 cases of unilateral nerve type deafness.

No. of Cases	0-9 Yrs.	10-19	20-29	30-39	40-49	50-59	60 and over	Male	Female	Rt. Ear	Lt. Ear
(Total 23)	2	5	2	5	6	3	0	11	12	8	15

deafness was sudden and acute and without any known related or precipitating factor.

GROUP II: *Sudden acute onset of deafness preceded by febrile illness.*

Four patients related a history of sudden acute hearing loss following an acute febrile episode, *e.g.* "grippe," and "flu."

GROUP III: *Deafness associated with recurrent episodes of tinnitus and severe vertigo with nausea and vomiting.*

Three patients gave histories of acute, severe recurrent episodes of tinnitus associated with vertigo, nausea and vomiting.

GROUP IV: *Total or sub-total deafness of unknown duration and cause.*

Six patients exhibited total or sub-total deafness that had existed for an indefinite period of time and without any known causative factor.

TABLE IV. CATEGORIES OF PATIENTS WITH UNILATERAL NERVE-TYPE DEAFNESS.

Case No.	Age	Sex	Onset	Precipating Cause	Tinnitis	Vertigo	Nausea & Vomiting	Audiometric Pattern
				GROUP I.				
7	24	M	Sudden	Unknown	+	Slight	0	Severe loss for all tones except low; *subsequent improvement;* BC markedly impaired; caloric tests normal and equal.
17	46	M	Sudden	Unknown	+	0	0	Subtotal deafness at onset; gradual *return* of low and high tones with middle sag.
20	42	M	Sudden	Unknown	+	0	0	Severe middle and high tone loss; *subsequent improvement* in middle tones; caloric tests equal and normal; BC good.
25	45	F	Sudden	Unknown	+	0	0	*Total deafness;* no caloric response on affected side.
36	12	F	Sudden	Unknown	+	+	+	Severe middle and high tone loss.
47	39	F	Sudden	Unknown	+	Slight	0	Severe middle and high tone loss; BC reduced.
52	48	F	Sudden	Unknown	+	+	+	Severe impairment for all tones; slightly better in low frequencies; BC reduced.
				GROUP II.				
8	38	F	Sudden	"grippe" with fever	+	0	0	Severe hearing loss for all tones; absent BC; severe attack of "*grippe*" shortly before onset of hearing loss.
18	18	F	Sudden	acute febrile episode	+	+	0	Severe middle and high tone loss; BC normal; onset right after *acute febrile* episode.
41	33	M	Sudden	acute febrile episode	+	0	0	Total deafness; followed "*scarlet fever*" at 14 years.
55	40	F	Sudden	"flu"	0	0	0	Loss for all frequencies; BC reduced; followed attack of "*flu*" and *fever* for 3 days.
				GROUP III.				
49	54	M	Recurrent	Ménière's Disease	+	+	+	Recurrent, violent episodes of rotatory dizziness with nausea; tinnitus very marked; audio shows flat type of curve with absent BC; intermittent for past 23 years.

50	34	F	Recurrent	Ménière's Disease	+	+	+	Episodic rotatory dizziness with nausea and vomiting over a 2 year period; flat audiometric curve with absent BC. Tinnitus marked.
51	41	M	Recurrent	Ménière's Disease	+	+	+	Two severe episodes of rotatory dizziness with nausea; marked tinnitus. Audiogram shows flat curve with absent BC.
				GROUP IV.				
6	19	F	Unknown	Unknown	0	0	0	Total deafness of one year's duration; no definite known cause; no tinnitus, no vertigo.
16	9	F	Unknown	Unknown	0	0	0	Total deafness of unknown duration; first observed by camp counsellor; no tinnitus, no vertigo; no known cause.
19	29	F	Unknown	Unknown	0	0	0	"Hearing loss" noted "all her life;" no known cause; no tinnitus, no vertigo; audiogram shows total deafness.
27	16	M	Unknown	Unknown	0	0	0	Hearing loss first apparent at about age of 10 years; no known cause; no tinnitus, no vertigo; audiogram shows sub-total deafness.
31	8	F	Unknown	Unknown	0	0	0	Hearing loss first apparent at about age of 4 or 5; no known cause; no tinnitus or vertigo; audiogram shows sub-total deafness.
54	15	F	Unknown	Unknown	0	0	0	Hearing loss first apparent about age of 4 or 5; no known cause; no tinnitus or vertigo; audiogram shows sub-total deafness.
				GROUP V.				
2	55	M	Slowly progressive	Unknown	+	0	0	Progressive hearing loss over a 3-year period; tinitus at onset which has persisted; no known cause; no vertigo; BC markedly reduced.
39	41	M	Slowly progressive	Unknown	0	0	0	Progressive hearing loss over a 3-year period; spontaneous without apparent cause; audiogram shows marked high-tone loss.
56	52	M	Slowly progressive	Unknown	0	0	0	Progressive hearing loss over a 4-year period; severe hearing loss with absent BC.

GROUP V: *Progressive deafness without apparent precipitating cause.*

Three patients exhibited gradually progressive deafness extending over a period of three to four years. No apparent cause could be ascertained to account for the hearing impairment.

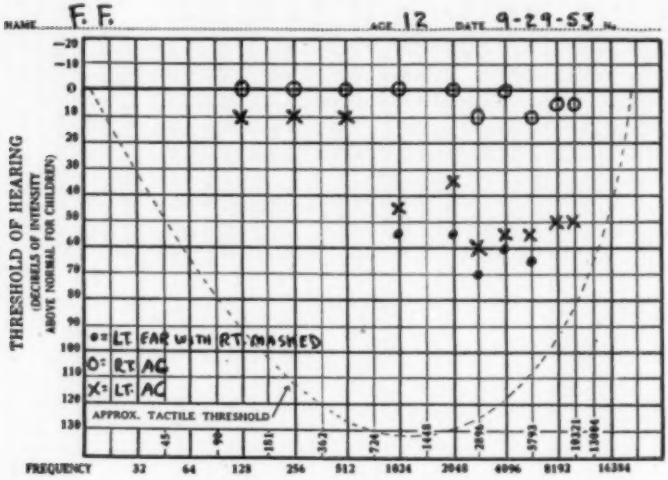

Fig. 4. Audiogram of patient with acute sudden onset of unilateral nerve type deafness without known precipitating factor.

ETIOLOGIC FACTORS IN UNILATERAL NERVE TYPE DEAFNESS.

In contrast to a single etiologic factor, *i.e.* otosclerosis, thought to be responsible for the 34 cases of unilateral conduction deafness, no one factor seemed to explain adequately the variety of manifestations exhibited by the 23 patients with unilateral nerve type deafness.

Examination of the five different groupings made of the nerve type deafness cases seems to offer some basis for speculation regarding etiologic factors for the patients in this series.

GROUP I. The factors common to each of the patients in this

group are: *a.* the abrupt onset of deafness; *b.* the absence of any apparent precipitating cause; and *c.* the presence of tinnitus. Two patients had vertigo with nausea and vomiting associated with the onset of deafness, while two patients complained of mild imbalance but without true vertigo or nausea. The remaining three patients in this group had no symptoms of vestibular disturbances. The youngest patient in this

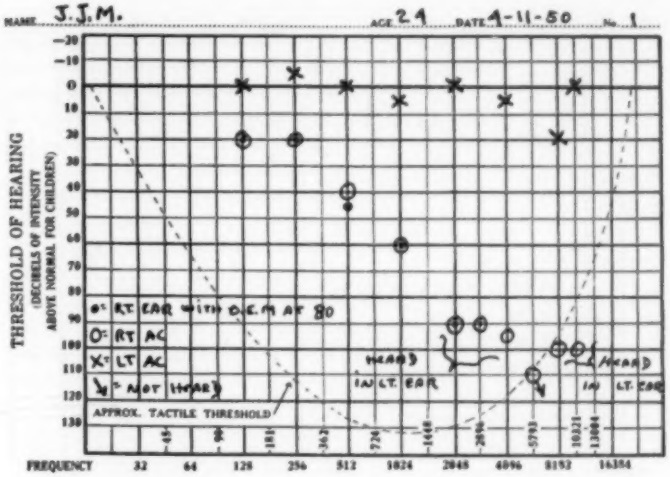

Fig. 5. Initial audiogram of patient with sudden acute unilateral nerve type hearing impairment of unknown etiology. (4 hours after onset of hearing loss.)

group was 12, and the oldest 48 years. Three patients were male, four were female.

The audiometric pattern in six of the seven patients was that of severe impairment for the middle and high frequencies with little or no involvement of the low frequency range (see Fig. 4). In one patient total deafness was present for all tones at the maximum intensity generated by the audiometer used. Cold caloric stimulation of the affected ear of this patient elicited no response; the opposite ear had normal responses.

Follow-up audiometric studies were made on three of the patients exhibiting middle and high frequency impairment. In all three patients, definite improvement in hearing for pure tone audiometry and for speech reception was observed. (See Figs. 5 through 15.)

GROUP II. The factors common to each of the four patients in this group were: *a.* abrupt onset of the deafness, and *b.* a

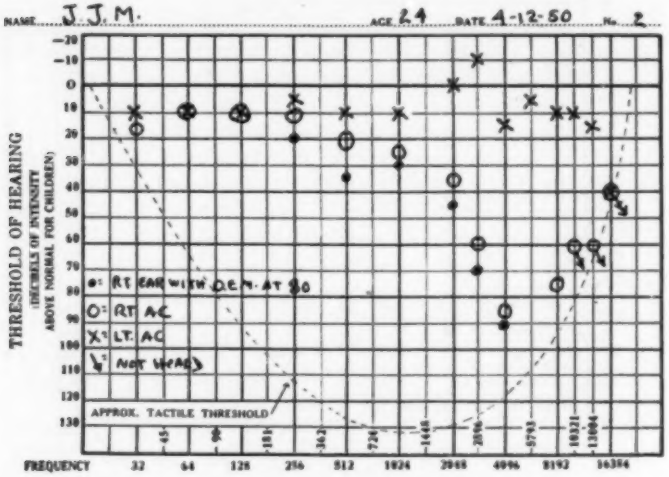

Fig. 6. Repeat audiogram showing hearing improvement in patient with sudden acute unilateral nerve type hearing impairment of unknown etiology. (24 hours after onset.)

history in each instance of an acute febrile illness, *e.g.* "grippe," and "flu," which preceded the hearing loss. Vestibular symptoms occurred in two patients while in the remaining two no disturbance of equilibrium was present. As in the patients in Group I in whom tinnitus was a constant symptom, three patients in Group II complained of tinnitus, while in the one remaining, tinnitus was absent.

The audiometric pattern observed in this group of patients differed somewhat from that found in patients in Group I. In one patient total deafness was present and was alleged to

have started following an attack of scarlet fever at the age of 14 years. In two patients severe hearing loss for all tones with reduced bone conduction reception was observed. In one patient severe middle and high tone loss was present (see Fig. 16).

GROUP III. The three patients who comprise this group all exhibited markedly similar symptoms of, *a.* abrupt, recurrent

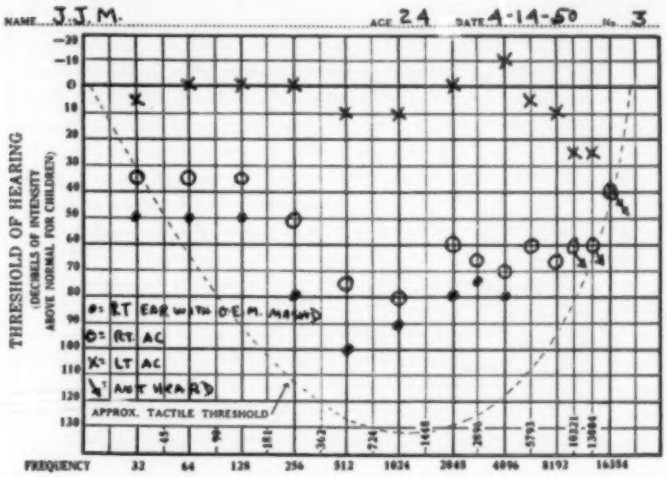

Fig. 7. Repeat audiogram showing hearing improvement in patient with sudden acute unilateral nerve type hearing impairment of unknown etiology. (3 days after onset.)

episodes of violent rotatory dizziness; *b.* nausea and vomiting associated with the dizziness; *c.* marked tinnitus and, *d.* a moderately severe hearing loss with a flat type of audiometric pattern and with absent bone conduction in the affected ear (see Fig. 17). It is felt that the basis of the symptoms is in each case an endolymphatic hydrops (Ménière's disease).

Although the patients making up Groups I, II, and III exhibited differences sufficient to permit classification into three different groups, nevertheless there remains a basic pattern common to all three groups. This consists of sudden

onset of severe hearing impairment in each case and with the exception of one patient, marked tinnitus associated with the hearing loss. The major differences in the three groups are, *a.* the absence of any apparent cause of the hearing loss in Group I; *b.* the history of acute febrile illness preceding the hearing loss in patients in Group II, and *c.* episodic character of the vestibular symptoms in patients in Group III.

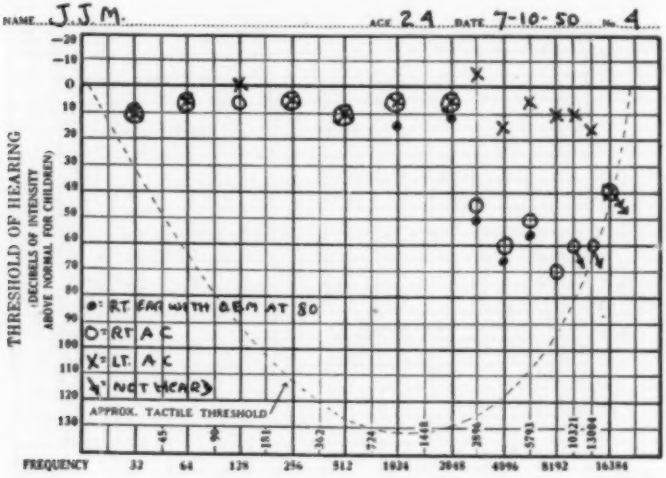

Fig. 8. Repeat audiogram showing hearing improvement in patient with sudden acute unilateral nerve type hearing impairment of unknown etiology. (3 months after onset.)

●=Rt. Ear with O.E.M. at 90 db.
O=Rt. AC.
X=Lt. AC.
✓=Not Heard.

It is difficult to ascribe a specific etiologic factor for the hearing loss observed in patients in Groups I and II. The histories and audiometric findings closely resemble patients described by Rasmussen,[7] and Lindsay and Zudema.[8] In these reports it was felt that a toxic-neuritis of the VIIIth cranial nerve was the probable causative factor.

The possibility of vascular disorders, *e.g.* spasm, thrombosis, or hemorrhage of the internal auditory artery or one of its

branches has been suggested as a possible cause for this type of deafness. Fowler[10] suggested that vascular spasm and concomitant blood sludging could produce symptoms similar to those manifested by patients in Group I and II. Hilger, J. A.,[17] advances a hypothesis that a disturbance of autonomic nerve control of the end arterioles of the labyrinth can produce vascular spasm. The resulting metabolic dis-

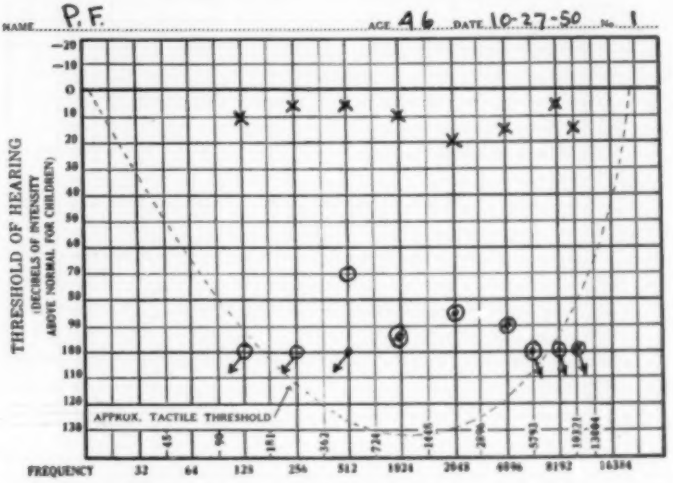

Fig. 9. Initial Audiogram of patient with sudden acute unilateral nerve type deafness of unknown etiology. (10 days after onset.)
●=Rt. AC with O.E.M. at 90 db.
X=Lt. AC.
↘=Not Heard.

turbances are capable of producing the clinical syndrome of sudden deafness and vertigo in the young individual in apparent good health.

Histologic preparations of the temporal bone were not available for any patient in this series, nor for any of the patients reported by the authors just cited; therefore, all of the suggested causes for the hearing loss must necessarily remain presumptive and speculative.

GROUP IV. The six patients in this group exhibited rather

striking uniform findings. All were under the age of 20 except one patient, who was 29 years old. Factors common to each patient in this group were: *a.* absence of any known cause or precipitating factor for the deafness; *b.* absence of any tinnitus; *c.* absence of vertigo; and *d.* sub-total or total deafness on the affected side.

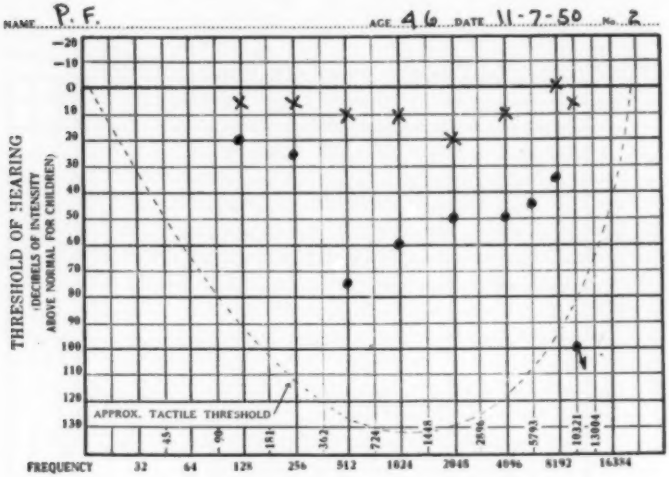

Fig. 10. Audiogram showing hearing improvement in patient with sudden acute unilateral nerve type deafness of unknown etiology. (21 days after onset.)

●=Rt. Ear with O.E.M. at 90 db.
X=Lt. AC.

The deafness which was present in this group of patients was the most severe of any group in the entire series. Three of the six patients exhibited total deafness, while in the remaining three only vestigial hearing could be determined.

The onset of the hearing loss occurred early in life for the group as a whole. One patient described her hearing loss as being present "all her life." In two patients, deafness was noted at about the age of four to five years; in one patient, at nine years of age. In the latter patient, the hearing impairment was first noted by the child's summer camp counsellor; the parents had not been aware of any loss (see Fig.

18). In one patient, 16 years of age, the onset of the hearing loss was thought to have begun at the age of ten years. The patient 19 years of age stated that the hearing loss allegedly began about one year previously.

This group of six patients presents a syndrome that differs markedly from the patients in Groups I, II and III. The involvement seems to affect only the auditory portion of the

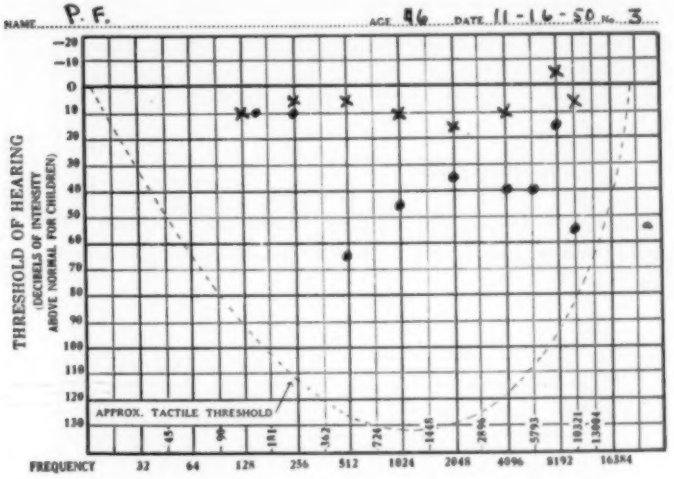

Fig. 11. Audiogram showing hearing improvement in patient with sudden acute unilateral nerve type deafness of unknown etiology. (30 days after onset.)

●=Rt. Ear with O.E.M. at 90 db.
X=Lt. AC.

VIIIth nerve, for in no patient was any vestibular disturbances noted.

Certain of the virus or ultra-virus diseases, such as mumps, have long been known for their specific neuro-toxic effect on the auditory portion of the VIIIth nerve. The occurrence of profound unilateral hearing loss without vestibular involvement following parotitis is a matter of established record.

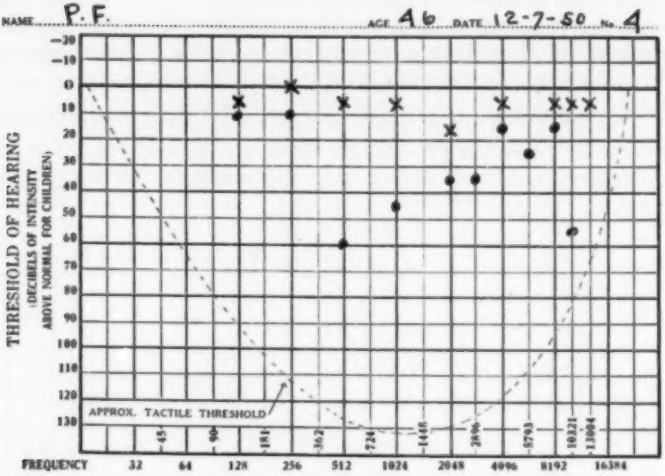
NAME P.F.
AGE 46 DATE 12-7-50 No. 4
THRESHOLD OF HEARING
(DECIBELS OF INTENSITY ABOVE NORMAL FOR CHILDREN)
APPROX. TACTILE THRESHOLD
FREQUENCY 32 64 128 256 512 1024 2048 4096 8192 16384

Fig. 12. Audiogram showing hearing improvement in patient with sudden acute unilateral nerve type deafness of unknown etiology. (51 days after onset.)

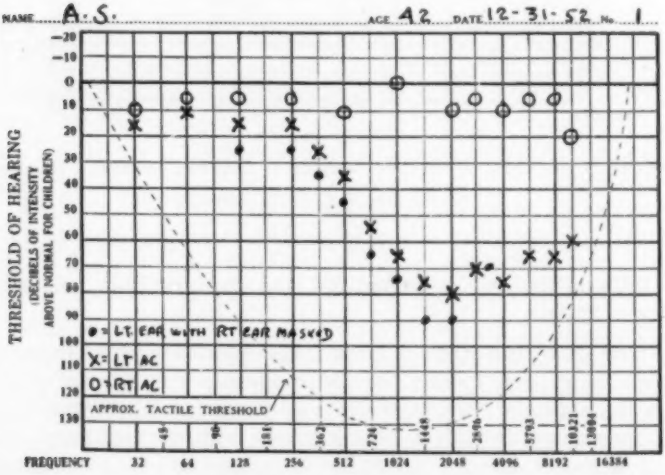
NAME A.S.
AGE 42 DATE 12-31-52 No. 1
THRESHOLD OF HEARING
(DECIBELS OF INTENSITY ABOVE NORMAL FOR CHILDREN)
⊙ = LT. EAR WITH RT EAR MASKED
X = LT AC
O = RT AC
APPROX. TACTILE THRESHOLD
FREQUENCY 32 64 128 256 512 1024 2048 4096 8192 16384

Fig. 13. Initial audiogram of patient with sudden acute unilateral nerve type deafness of unknown etiology. (8 days after onset.)

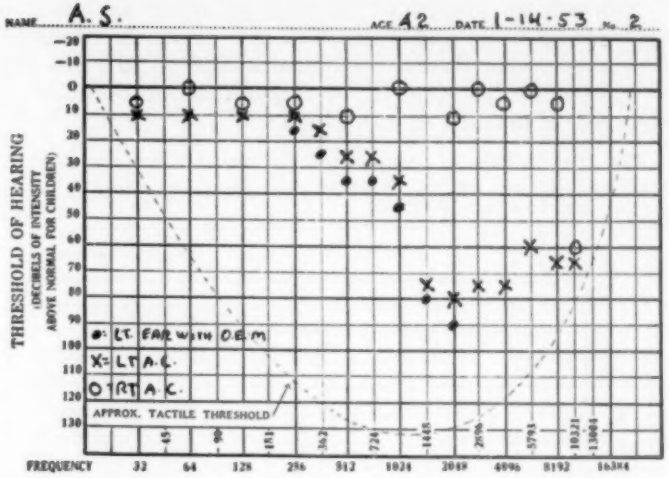
NAME A. S.
AGE 42 DATE 1-14-53 No. 2
THRESHOLD OF HEARING
(DECIBELS OF INTENSITY ABOVE NORMAL FOR CHILDREN)
●: LT. EAR WITH O.E.M.
X: LT A.C.
O: RT A.C.
APPROX. TACTILE THRESHOLD
FREQUENCY 32 64 128 256 512 1024 2048 4096 8192 16384

Fig. 14. Audiogram showing hearing improvement in patient with sudden acute unilateral nerve type deafness of unknown etiology. (22 days after onset.)

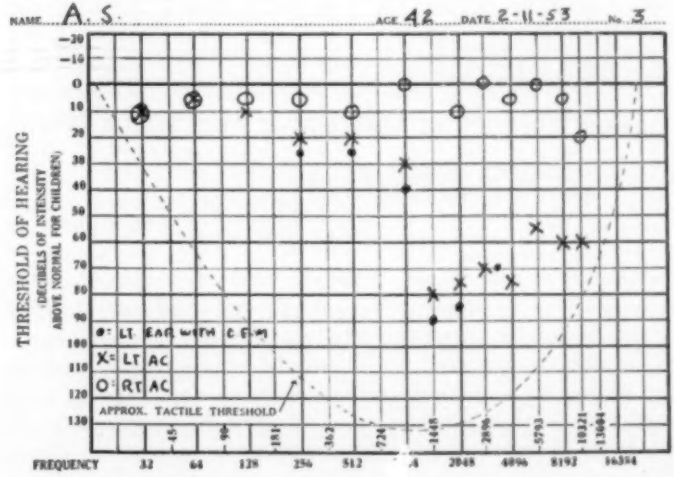
NAME A. S.
AGE 42 DATE 2-11-53 No. 3
THRESHOLD OF HEARING
(DECIBELS OF INTENSITY ABOVE NORMAL FOR CHILDREN)
●: LT EAR WITH O.E.M.
X: LT AC
O: RT AC
APPROX. TACTILE THRESHOLD
FREQUENCY 32 64 128 256 512 1024 2048 4096 8192 16384

Fig. 15. Audiogram showing hearing improvement with sudden acute unilateral nerve type deafness of unknown etiology. (50 days after onset.)

Although no history of deafness following an attack of epidemic parotitis was obtained in any patient in Group IV, it is conceivable that a subclinical attack of parotitis may have been the etiologic factor responsible for the deafness observed in patients in this group.

The possibility of congenital failure of development or maldevelopment of the auditory mechanism must also be

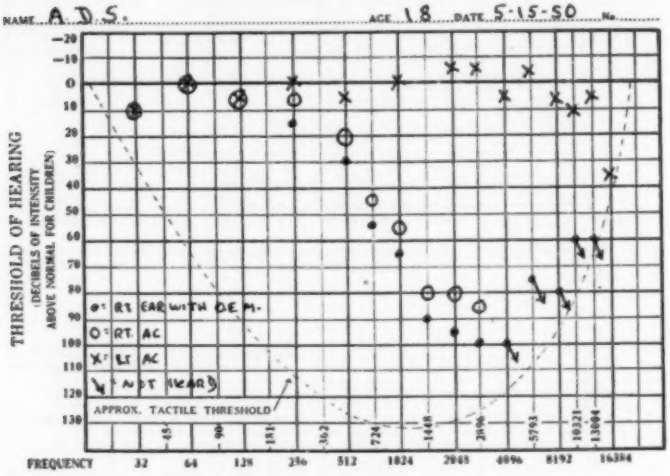

Fig. 16. Audiogram of patient with sudden acute unilateral nerve type deafness following an acute febrile illness.
Weber (512 D-V Steel Fork): Referred to right ear.
Rinne (512 D-V Steel Fork): AC>BC { AC=60-65 Sec. (Normal Duration) / BC=30-35 Sec. (Normal Duration)
Lt. AC>BC { AC=15-20 Sec. / BC=0

considered in attempting to explain the deafness observed in this group of patients. The relationship between maternal rubella in the first three months of pregnancy and the appearance of embryonic defects in the infant, among them deafness, is well established, and may be responsible for the type of impaired hearing observed in patients in Group IV.

Other factors which may produce a congenital type of

hearing defect include hereditary deafness, Rh factor, toxemia of pregnancy, congenital deformities of the auditory apparatus, immunization during pregnancy, birth injuries as result of obstetric difficulties or abnormalities, cerebral degeneration, anoxia or cyanosis after birth. While none of the foregoing causes were known to have occurred in patients in

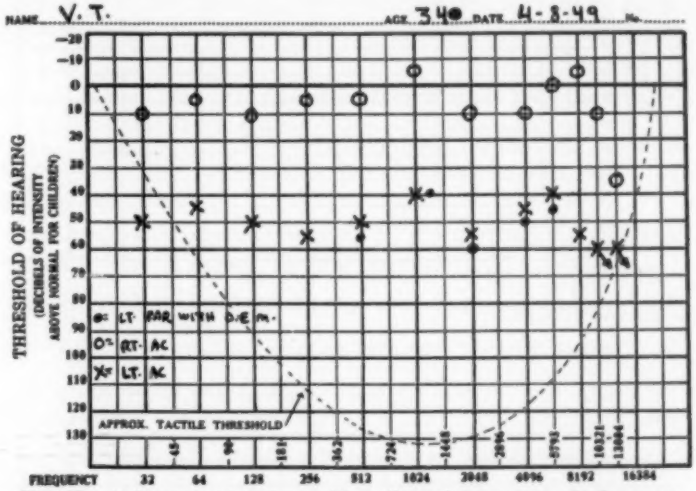

Fig. 17. Audiogram of patient with unilateral nerve type deafness associated with acute, severe episodic vertigo with nausea, vomiting and tinnitus.

O=Right AC.
X=Left AC without masking of right ear.
Note "shadow curve" effect.
●=Left ear with right masked at 85 db.
↙=Not Heard.

Group IV, their possible etiologic role in the deafness observed cannot be excluded.

GROUP V. The three patients in this group exhibited hearing loss characterized by, *a.* progressive impairment over a three to four year period, and *b.* absence of any known related or precipitating factor. All three patients were males and all were beyond the age of 40 (see Fig. 19). The type of hearing impairment observed in these three patients is similar to that in certain patients described by Crowe, S. J.,[18]

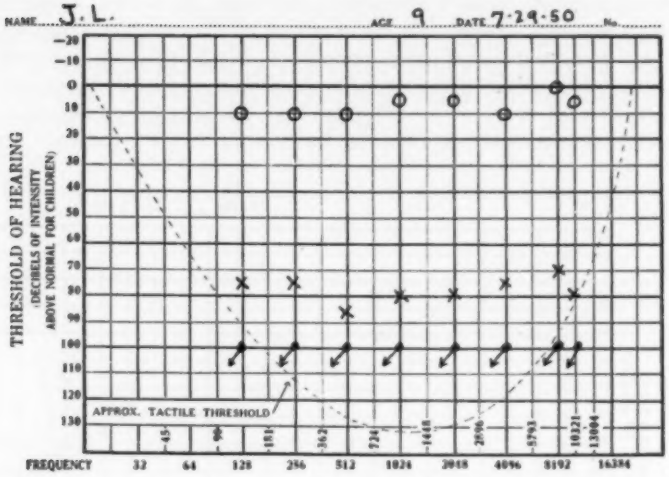
NAME J. L. AGE 9 DATE 7-29-50 No.
THRESHOLD OF HEARING
(DECIBELS OF INTENSITY ABOVE NORMAL FOR CHILDREN)
APPROX. TACTILE THRESHOLD
FREQUENCY 32 64 128 256 512 1024 2048 4096 8192 16384

Fig. 18. Audiogram of patient with unilateral total deafness of unknown origin and duration.

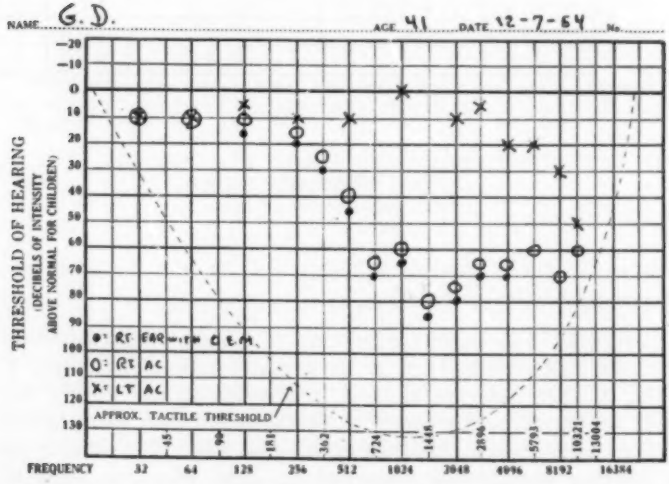
NAME G. D. AGE 41 DATE 12-7-64 No.
THRESHOLD OF HEARING
(DECIBELS OF INTENSITY ABOVE NORMAL FOR CHILDREN)
APPROX. TACTILE THRESHOLD
FREQUENCY 32 64 128 256 512 1024 2048 4096 8192 16384

Fig. 19. Audiogram of patient with gradually progressive unilateral nerve type hearing impairment.

et al., in their study on the histopathology of high tone deafness. These authors, in their histologic material, demonstrated certain anatomic changes, specifically atrophy of the organ of Corti and nerve, stria vascularis and sulcus cells. Although no reference to progression of hearing loss was recorded in their study, it is suggested that similar histopathologic changes of neural elements of the cochlea may account for the hearing loss observed in the above three patients.

SUMMARY AND CONCLUSIONS.

1. A study of 57 patients exhibiting unilateral deafness was made. For inclusion in the study group, the following criteria were used:

a. Presence of normal hearing in one ear and hearing loss ranging from moderate to total deafness in the other ear.

b. Normal findings in both ears on otoscopic examination.

c. Absence of otitis media or sequelae of otitis media.

d. Absence of significant abnormalities of the upper air passages and nasopharynx.

e. Exclusion of acoustic trauma, intracranial neoplasm and head injuries as possible causative factors in the unilateral hearing loss.

2. In a series of 1475 patients with hearing impairments of all types, 57 patients met the above criteria, an incidence of 3.8 per cent.

3 Unilateral conduction type deafness was observed in 34 of the toal of 57 patients and unilateral nerve type deafness in 23.

4. Histologic preparations of the temporal bone were not available for any patient in this study, so that suggested etiologic causes are presumptive.

5. In 34 patients with unilateral conduction deafness, it is felt that otosclerosis is the probable basis for the deafness in each case.

6. In 23 patients with unilateral nerve type deafness, the probable causative factors of the deafness vary considerably:

a. In 11 patients the suggested causes for the deafness include: *1.* Toxic neuritis of the VIIIth nerve, *2.* Vascular disorder of the internal auditory artery, and *3.* Disturbance of the autonomic nerve control of the end arterioles of the labyrinth.

b. In three patients, it is felt that the basis of the symptoms is an endolymphatic hydrops (Ménière's disease).

c. In six patients the cause of the hearing loss is quite obscure. Subclinical mumps, congenital mal-development of the auditory mechanism, and maternal rubella are suggested as possible causes for the deafness; in others less common causes include toxemia of pregnancy, hereditary deafness and birth injuries.

d. In three patients the observed hearing loss is thought to be due to degenerative changes of neural elements of the cochlea.

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TWO FELLOWSHIPS TO RECRUIT WORKERS IN AUDIOLOGY AND SPEECH.

Establishment of two fellowships in Audiology and Speech at Northwestern University is announced by the American Hearing Society, its member agency, the St. Paul Hearing Society, and the university. The new training program will be geared to preparing qualified persons for work in local hearing societies.

The fellowships, made possible by a bequest from the Myers Foundation of St. Paul, Minn., are to be known as The Reine Humbird Myers Fellowships. Each fellowship will pay $3,000 a year for two years.

Applicants may write to Crayton Walker, executive director, American Hearing Society, 1800 H St., N. W., Washington 6, D. C., or to Dr. Raymond Carhart, School of Speech, Northwestern University, Evanston, Ill.

MODALITY OF THE COCHLEAR HUMORAL CIRCULATION.*†

PROF. E. BORGHESAN,

Palermo, Italy.

Despite the numerous experimental researches, it has not been possible to demonstrate the places and the modality of production, or absorption of the cochlear fluids, and the direction of their motions.

From Corti to date, the cochlear endolymph has always been considered to be secreted by the *stria vascularis*, so that Retzius has compared it to the *tegmentum vasculosum* of birds, which is known to secrete endolymph.

Some researchers held that the stria is not the only source of the cochlear endolymph. According to Halle, Rudinger, etc., it is also derived from the cerebrospinal liquid. Gegenbauer, Siebermann, Quix, Egmond, and others, think that the perilymph provides a part of the endolymph; Saxen believes that it is derived from the stria, the cerebrospinal liquid and from the perilymph.

I emphasize that Shambaugh, who described the epithelial cells (1908) that occur in the outer spiral sulcus and proceed into the connective tissue of the ligament, suggested that the endolymph might be secreted, in addition to the stria, by the outer spiral sulcus.

In a recent study, Lawrence held that in the spiral prominence *rosette cells* are found, which provide the nutrient material, while the stria provides a constant quantity of endolymph.

According to the author's idea, the stria cannot secrete endolymph, because its histological structure is inadequate.

All who have studied this problem agree that the produc-

*Read at the meeting of the Sixth International Congress of Otolaryngology, Washington, D. C., May 5-10, 1957.

†From the Oto-Rhino-Laryngological Clinic of the University of Palermo.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication May 15, 1957.

tion of the endolymph is strictly controlled and automatically regulated to avoid variations in the endolymph tension; the hypotheses advanced about the place and the modality of functioning of the regulating device vary and are not always convincing.

In my opinion, an arrangement exists which regulates the production of the cochlear endolymph. It avoids variations in the tension and provides oxygen and other nutrient material to the hair cells, according to their activity.

The site of resorption of the cochlear endolymph is uncertain; the experiments and the hypotheses concerning it suggest the endolymphatic sac (Guild), the outer spiral sulcus (Quix and Egmond, Kley, Altmann and Waltner, Saxén), and many other sources (Lindsay).

The uncertainty of the site of production and absorption of the cochlear endolymph depend also upon the direction of the endolymphatic stream. If we accept the places presumed to be responsible for both functions, according to the various hypotheses, there would issue either a direction neglecting the organ of Corti, or there would be a mixing between the endolymph rich in catabolytes and that freshly produced. In both cases the assumption concerning the origin and absorption of this fluid, leaves some other problems to solve, for instance, that of the multiplicity of the regulating mechanism; besides, it would be difficult to understand why the organ of Corti should be nourished by fluids that have already fulfilled other functions.

In 1948, after a study of the structure of the tectorial membrane and its relationship to the organ of Corti, it was necessary to extend the histological researches to the spiral ligament, the prominence and the limbus. I carried out some histological investigations in rabbits, guinea-pigs and the human fetus; the results were described in the literature from 1949 to 1953. Now I refer to them in synthesis and I think it possible, with our increased knowledge of the morphology and topography of the anatomical structures, to suggest an hypothesis about the modality of the cochlear humoral circulation.

SPIRAL LIGAMENT—SPIRAL CANALICULAR SYSTEM.

The fibers of the connective tissue, constituting the spiral ligament, directed towards the basilar membrane, end in different ways: on the side of the scala tympani, the fibrils reach the basilar membrane, while those from the side of the outer spiral sulcus, form little bundles, separated by clear, but more or less wide spaces. The fibrils constituting the more posterior of these bundles, insert themselves in the substance of the outer spiral sulcus, where they are covered by epithelial cells (see Fig. 1). Other fibrils, placed between these bundles and the spiral prominence, are grouped in a rostrum-like manner, ending at some distance from the cells of the outer spiral sulcus, so that between these latter fibrils and the terminal part of the rostra, a large cavity exists where the united spaces are interposed between the rostra (see Fig. 2). All these cavities, filled with epithelial cells probably derived from the cells of the outer spiral sulcus, form a *spiral canalicular system.* This system which is barely noticeable in rabbits, is found to be somewhat different in guinea-pigs and in human fetus.

SPIRAL PROMINENCE—SPIRAL VILLI.

When seen in a transverse section, the spiral prominence is oval; in some specimens it appears to be composed of connective tissue, partially covered with epithelial cells, which seem to spring from the stria, dividing into two strata (see Fig. 1). The deeper cellular stratum partially covers the connective tissue, while the more superficial stratum separates the prominence from the cochlear duct, giving rise to a more or less evident space. When seen in a spiral projection, the prominence appears constituted of fibro-epithelial formations, close to one another (see Figs. 1-3) and surrounded by a clear space. The connections of the capillaries, with the cells, are seen in Fig. 2. The spiral prominence seems, therefore, to be composed of villi, each of them, for a more or less extended area project into a cavity communicating (see Fig. 2) with the spiral canalicular system, while it is separated from the cochlear duct by a layer of cubic or flattened cells.

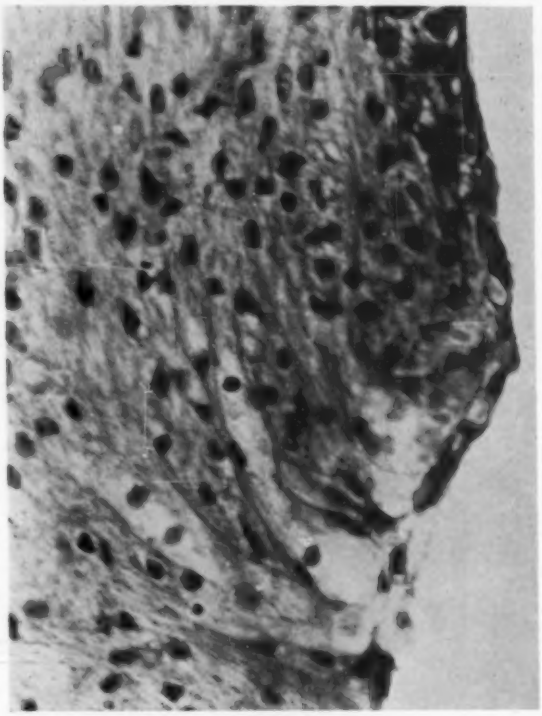

Fig. 1. Transverse section of spiral ligament. Bundles of connective tissue separated by spaces filled with cells; the more anterior one is in relationship with the spiral prominence; all the spaces unite in a cavity near the outer spiral sulcus. The whole of these spaces forms the spiral canalicular system. Rabbit.

The epithelial cells of the prominence are special. Their nuclei have the marginal chromatin in the form of a dotted ring. In some the nucleus (see Fig. 3) appears to be in mitosis, in the phase of the equatorial plate. The protoplasm of these cells is voluminous and shows some filaments, which start from the nucleus and extend toward the protoplasmatic membrane; in other projections the protoplasm is granular.

STRIA VASCULARIS.

Nothing is to be added or modified in Saxén and Fieandt's

Fig. 2. The differentiated epithelial cells of the prominence are in connection with a capillary seen in cross section. Their protoplasm shows filaments, while the nucleus is ring-shaped. Guinea-pig.

description. It is only to be noted that the stria does not contain the clear cells, which in the cochlear duct transfer fluid.

VASCULARITY OF THE SPIRAL LIGAMENT.

Both the arterial and the venous vessels of the spiral ligament morphologically appear alike and seem to be capillaries. The arterial system starts from the anterior branch of the spiral artery, which sends two or three branches to the stria, one or two to the prominence (see Fig. 4), and finally bifucates in terminal branches; one communicates with a

vein reaching the scala tympani, forming Agazzi's arterovenous arch; the other goes to the outer spiral sulcus, branching out in the spiral canalicular system, where it forms a series of loops, that lie in the substance of the outer spiral sulcus and come into contact with the walls of the canalicular system and with the cells of the outer spiral sulcus (see Figs. 4, 5, 6).

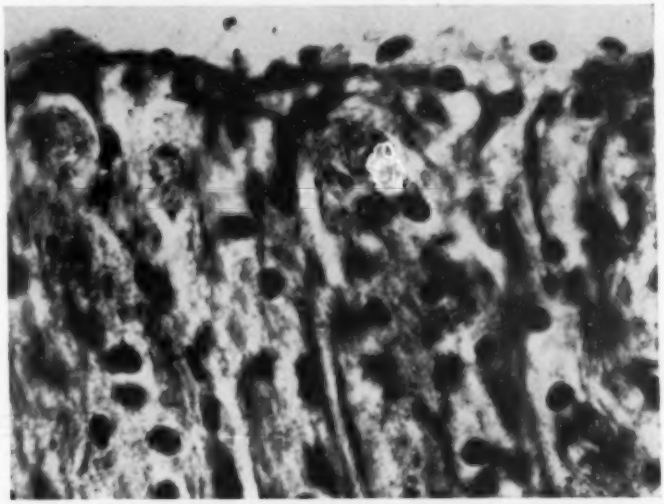

Fig. 3. The prominence is formed by villi; each is surrounded by a cavity separated by the cochlear duct. The epithelial cells of the villi are polymorphous; the protoplasm is dotted and in the nucleus the chromatin is marginal. Rabbit.

The veins of the stria reach the prominence (see Fig. 4).

In addition to the arterial basin, the spiral canalicular system contains a venous basin formed by vessels originating in the prominence (see Figs. 4, 5, 6). The loops of capillaries constituting the venous basin are placed perpendicularly to those of the arterial. The blood gathers in big veins going toward the scala tympani.

HAIR CELLS AND TECTORIAL MEMBRANE.

Concerning the hair cells, in the author's observations in

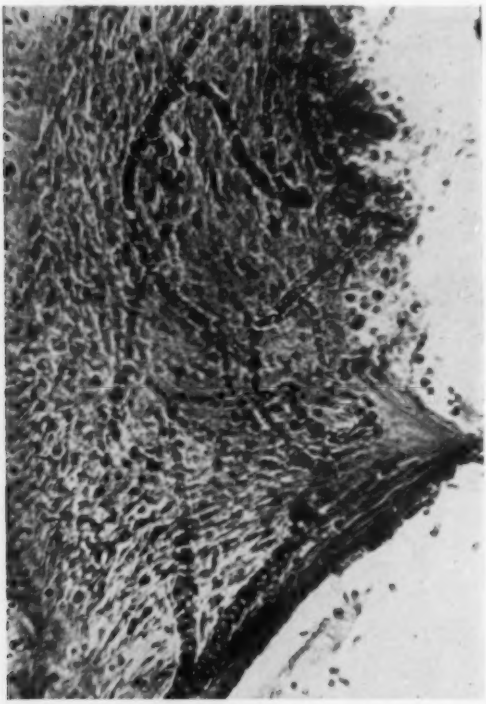

Fig. 4. Artery of the ligament branching out to the prominence; it ends bifurcating: one branch forms the artero-venous arch and the other rotates the outer spiral sulcus, forming loops on the jelloid stratum. Veins going from the stria to he prominence; from there, other veins go to the outer spiral sulcus. Guinea-pig.

preceding studies, which may prove of considerable importance with regard to the cochlear humoral circulation, these cells are supposed to be provided with very long cilia (see Figs. 7-8), composed of two segments, one, the basillar article, is well-known, being the characteristic cilium; the other segment is the flagellum, which I considered as a resonator.

The flagelli, surrounded by an amorphous cuticular substance, then form the tectorial membrane. The point of union of the two segments, flagello-basilar junction, is most fragile

and breaks quite easily, so that tectorial membrane and organ of Corti almost always appear separated.

In the author's opinion, therefore, the tectorial membrane must be regarded as an integral, constitutional part of the organ of Corti, *i.e.*, a unique anatomical and functional entity.

The tectorial membrane is inserted into the limbus and in the protoplasmatic membrane of Hensen's cells; also, it is joined to the organ of Corti in the vicinity of Claudius' inner

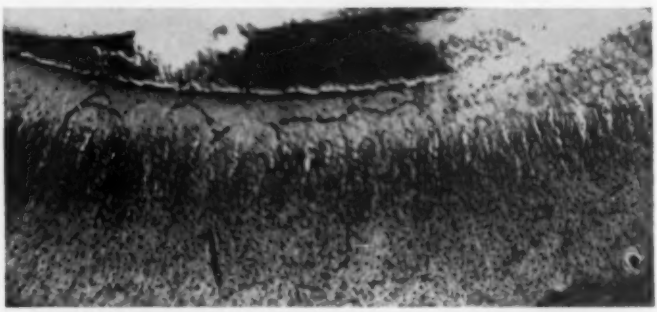

Fig. 5. Loops of capillaries surround the cavities of the canalicular spiral system and reach the jelloid layer of the outer spiral sulcus and are covered with epithelial cells. Rabbit.

cells and, what is very important from the functional point of view, to the hair cells (see Figs. 7-8).

According to Wittmaak, the organ of Corti and tectorial membrane are united by means of cuticular bridges. This is denied by others. In the author's opinion, the bridges do exist (see Fig. 7), and are formed by the basilar portions of the cilia, surrounded by the cuticular substance of the tectorial membrane (see Figs. 7-8). Every hair cell is united to the tectorial membrane by means of a pillar, containing cilia, the *hair pillar*.

The hair cells are found in the organ of Corti in a characteristic manner, so that when seen from its anterior face it appears as a chess-board. This aspect is due to the fact that the hair cells are interposed with the phalanges of Deiters' cells.

Each hair pillar is separated from the other by a space, corresponding to the phalanx, and a much larger space, corresponding to the heads of Corti's pillars. All of these spaces are limited by the phalanges, by the tectorial membrane and by the hair pillars, forming a *subtectorial canalicular net* (see Figs. 8-11).

This is closed from the outside by the insertion of the tectorial membrane in Hensen's cells and from the inside by

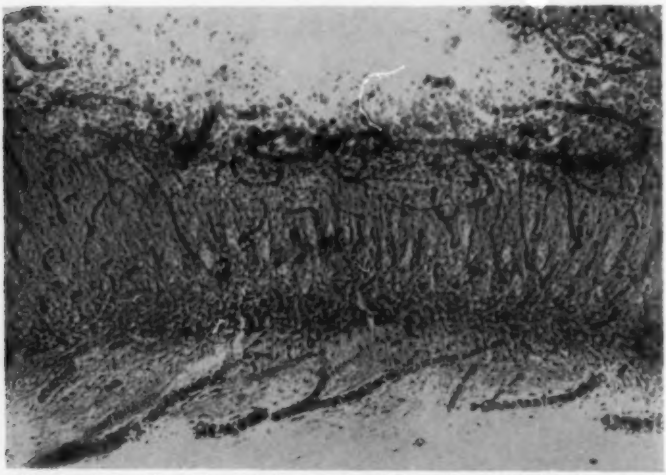

Fig. 6. Frontal section of the outer spiral sulcus. Capillaries coming from the spiral prominence form loops in the canalicular spiral system. Guinea-pig.

its insertion into the Claudius' inner cells. The union of the tectorial membrane to the organ of Corti, signifies that, in the living, the inner spiral sulcus is a canal (see Figs. 7-11).

LIMBUS.

In the second turn of the cochlear duct, the limbus of rabbits, in a cross section (see Fig. 9), presents a central cribriform zone, due to the cross section of many capillaries running in the loose connective tissue (see Figs. 9-10). Like the outer spiral sulcus, the basilar membrane, the inner spiral

sulcus, and the interdental furrows are lined with jelloid substance. These communicate with the connective tissue by means of fissures. This is better observed in the human fetus (see Fig. 10) in which, within the interdental furrows, capillaries are often found.

Corresponding to the insertion of Reissner's membrane, a clear zone is found (see Fig. 9), bathed by perilymph, which sometimes proceeds into the scala vestibuli. The numerous

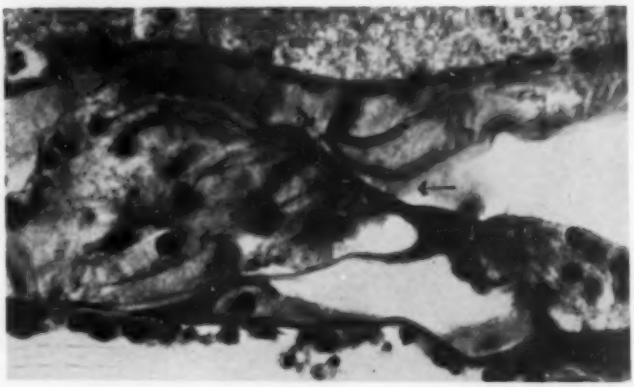

Fig. 7. Connections between organ of Corti and tectorial membrane. The arrows indicate the points of union of the two structures, constituted by the basilar articles of the cilia, surrounded by the cuticula (hair pillar). Guinea-pig.

capillaries of this zone seem lined with epithelial cells. These latter have polymorphous protoplasm; sometimes polyhedral, gray or clear; the nucleus is large, either central or eccentric. On account of its structure I called this tract of limbus the *vasculo-epithelial zone*.

The veins of the limbus originate from the medial branch of the spiral artery and run toward the inner spiral sulcus and the edge of the bony spiral lamina. Capillaries, after crossing the vasculo-epithelial zone, form loops near the interdental furrows; other capillaries enter the bony spiral lamina, form large loops and then reach the efferent branches of

Rosenthal's canal, where they run along with the nervous fibrils. Occasionally in the human fetus, the veins form loops along the basilar membrane; in rabbits a capillary, adherent to the anterior face of Reissner's membrane, from the spiral ligament reaches the vasculo-epithelial zone.

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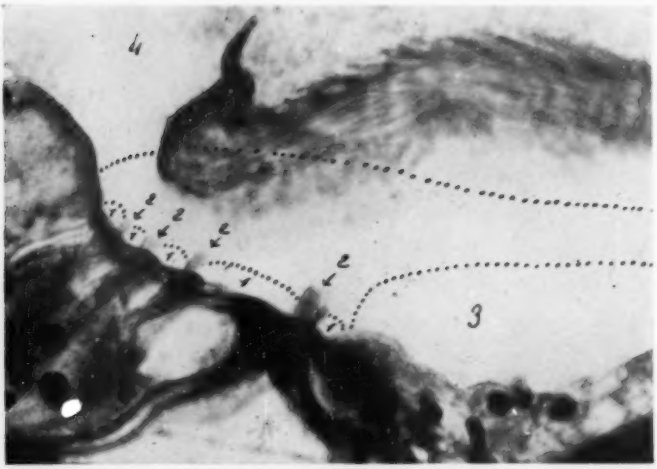

Fig. 8. Organ of Corti with hair (hair pillars) and detached tectorial membrane. The dotted line indicates the connections of the tectorial membrane with the organ of Corti in the living. 1—spaces of the canalicular subtectorial net; 2—hair pillars; 3—inner spiral duct; 4—cochlear duct. Rabbit.

The schematic reconstruction of the cochlear duct (see Fig. 11), according to the well-known elements and those which I have described, shows that the true *cochlear duct* and the *inner spiral duct* are the two largest cavities. The first cavity is limited by Reissner's membrane, by the anterior face of the limbus, the tectorial membrane, the organ of Corti, by the outer spiral sulcus, and by the prominence and the stria vascularis.

The inner spiral duct is limited by the outer face of the

limbus, the bony spiral lamina, the organ of Corti and the posterior face of the tectorial membrane.

These two wide endolymphatic spaces are separated and at the same time connected, by the tectorial membrane and by the organ of Corti. In addition, it appears evident that this membrane is the *only* cochlear formation *not covered* with epithelial cells, bathed by endolymph.

Fig. 9. Limbus of rabbit. 1—cribrous zone; 2—vasculo-epithelial zone.

These findings, I think, acquire great importance, both with regard to the valuation of the cochlear humoral circulation and to the modality of stimulation of the hair cells.

We are not particularly interested in this latter question, which has been the subject of some papers by this author.

The new anatomical elements allow us to sustain the fact that the stria vascularis of mammalia cannot be compared with the *tegmentum vasculosum* of birds, because the latter is the equivalent of the complex apparatus existing in the outer wall of the cochlear duct of mammalia: the stria vascularis, the villi of the spiral prominence, the spiral canalicular system

and the cells of the outer spiral sulcus. Only in this manner does the source of the cochlear endolymph of mammalia appear to be provided with the structures necessary to perform the same function as the tegmentum vasculosum.

Were this not true, it would be absurd to say that the cochlear duct of birds, rudimentally compared with that of the mammalia, might be endowed with an endolymph-producing

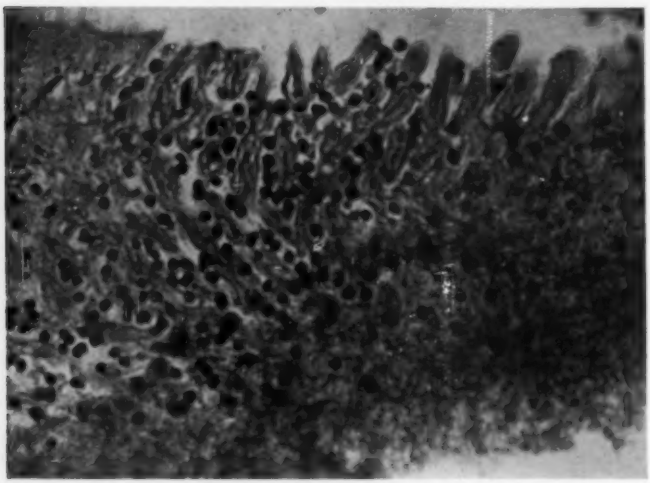

Fig. 10. Interdental furrows continuing in the loose connective tissue, where a vessel can be seen. Human fetus of six months.

apparatus more complete and more highly developed than that of the mammalia.

I believe that this delicate and complex function of the safety and regulating device, is undertaken by the spiral canalicular system, with a modality both simple and efficient, because morphology and structure utilize the direction of fluids across the cellular membranes.

As I suggested, the modality of the cochlear endolymphatic secretion repeats the renal one; therefore, each structure of the cochlear endolymph-secreting apparatus would have its

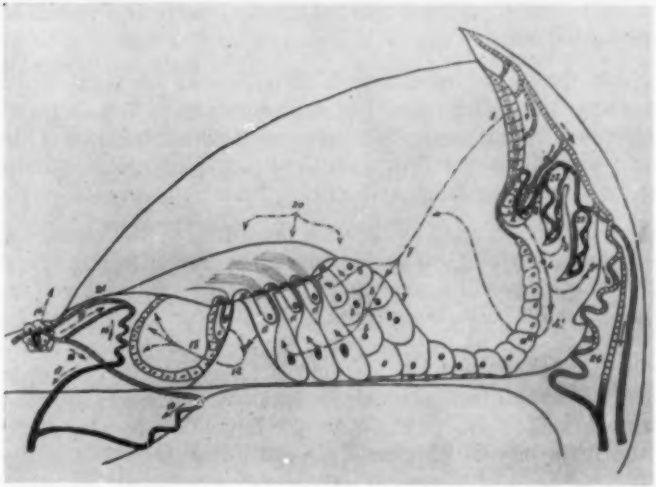

Fig. 11. Scheme of the structure of the cochlear duct and of the endolymphatic circulation. 1—Stria vascularis secreting crystalloids; 2—Villus forming the spiral prominence; 3-4-5—canalicular spiral system, where gathers the plasma secreted by the villi;- 6—cells of the outer spiral sulcus regulating the amount of plasma to be transferred into the cochlear duct; 7—endolymph formed by the secretions of the stria and of the villi; 8—Hensen's cells transferring nutrient liquid to Deiters' and to the outer hair cells; 9—Hensen's cells transferring nutrient liquid into the 10—subtectorial canalicular net, that transmits it towards the inner hair cells; 11—Nuel's spaces gathering waste liquid; 12—tunnel of Corti, waste liquid collector duct; 13—inner spiral duct transferring towards the interdental furrows the waste liquid to be absorbed; 14—vasculo-epithelial zone secreting perilymph; 15-16-17-18-19—vascular disposition of the limbus, absorbing liquid; 20—anterior face of the tectorial membrane placed in position optima to receive the sound pressures; 21-place of absorption of the endolymph that has the acoustic function; 22-23—venous and arterial basins of the canalicular spiral system that absorb plasma secreted in excess; 24—artero-venous arch, towards which the arterial stream deviates in case of plasma depletion from the villi. In the outer wall of the cochlear duct can be found the cochlear endolymph-producing apparatus and the safety-device.

own particular activity. I am convinced that this unsuspected subdivision of work may be the reason for which the stria has been considered inadequate to secrete the necessary quantity of endolymph, and to require the aid of the perilymph and of the cerebrospinal liquid, as efforts have been made to demonstrate.

According to the results of the morphological and cytological interpretation of the different structures, the stria would not secrete endolymph, but instead only its crystalloids;

the villi would perform the function of secreting the endolymphatic plasma.

Such functional modality, I think, must serve a double purpose: the strict control of the secretion of the different elements forming the endolymph and the maintenance of the safety-device by osmosis, filtration, and diffusion, besides the action of the mechanical and hydrostatic pressures.

The plasma secreted by the villi, according to the interpretation of the histological specimens, is poured into the spaces surrounding the villi; namely, in the spiral canalicular system. The epithelial cells, here contained, transfer the plasma towards the cells of the outer spiral sulcus; according to the need, regulated on the contingent activity of the hair cells, it then flows into the cochlear duct, were the crystalloids, secreted by the stria, go directly. Endolymph is formed at the moment the crystalloids and plasma unite.

That part of endolymphatic plasma that the cells of the outer spiral sulcus do not transfer into the cochlear duct would be resorbed into the venous and into the arterial basins. The former circulates blood derived from the villi, dehydrated for yielding plasma and, therefore, in the best condition for resorbing it. The arterial basin would start its activity in case of plasma depletion from the villi, as in vasomotor disorders; in this case the arterial blood, normally circulating in its basin, would be deviated towards the artero-venous arches.

In the spiral ligament, therefore, is found an arrangement to absorb liquid: not the endolymph, but a superfluous endolymphatic plasma.

The cells of the outer spiral sulcus may transfer fluid only from the canalicular system towards the cochlear duct, and not in the opposite direction.

It should be remembered that a thin layer of jelloid substance extends from the spiral canalicular system to the loose connective tissue of the limbus; as though Nature aimed at making the posterior wall of the cochlear duct impermeable.

Once it has been decided that the outer spiral sulcus does

not absorb endolymph, attention must be placed on the limbus, whose interdental furrows seem to constitute formations apt to convey and absorb it.

This author contends that the endolymph, after reaching the interdental furrows, must be transferred from the epithelial cells occupying these furrows into the underlying connective tissue and then carried, by means of the capillaries, into the venous stream.

The above-mentioned places of production and absorption of the cochlear endolymph, suggest that the direction of the endolymphatic stream is radial; in the cochlear duct, at any rate, everything tends to confirm this deduction.

In Fig. 11 it is evident that the organ of Corti and the tectorial membrane form a septum, separating the two great endolymphatic spaces. In the former is found the endolymph-producing apparatus; in the latter, the site of absorption; consequently, endolymph in order to pass from one compartment into another must circulate in the organ of Corti, carrying oxygen and other elements to it and relieving it from catabolytes.

It is well known that endolymph circulates in the organ of Corti to nourish it (Wittmaak, Mygind, Kobrak, etc.); besides, endolymph transmits sound pressures stimulating the hair cells. The morphology and the topography of every element forming the neuro-sensorial part of the phono-receptor apparatus, indicates that endolymph performs these two functions separately, almost as though the one should not interfere with the other, although they are strictly interdependent.

The endolymph that circulates in the organ of Corti and nourishes it has no reason to be subjected to the pressures and, in fact, this is not true, while the part which has no nutrient function, except that of transmitting the pressures, does not circulate in the organ of Corti, but is in direct relation with the anterior face of the tectorial membrane.

This, according to my opinion, is most suggestive, both regarding the cochlear humoral circulation and the modality of stimulation of the hair cells.

I believe that endolymph runs in the organ of Corti in the following manner: Hensen's cells absorb endolymph from the true cochlear duct to transfer it toward Deiters' cells and toward the subtectorial canalicular net; this conveys the endolymph towards the inner hair cells, while Deiters' cells transfer it to the outer cells. The endolymph, coming from all the hair cells and containing catabolytes, would be poured into Corti's tunnel, that assumes the function of a duct collecting waste endolymph. From the tunnel it flows into the inner spiral duct, and is absorbed through the interdental furrows of the outer face of the limbus.

The part of endolymph bathing the anterior face of the tectorial membrane would be absorbed in the limbus, by means of the interdental furrows of its anterior face.

The sound pressures, I believe, are transmitted to the anterior face of the tectorial membrane, making the flagelli resonate. The reason why this membrane is the only cochlear formation bathed by endolymph, but not lined with epithelial cells, is to facilitate to the greatest degree, the propagation of the sound pressures toward the flagelli.

The intralabyrinthine system of conduction of sounds, as I have noted elsewhere, ends with the flagelli, while that of the reception of sounds begins in the basilar articles, where, in my opinion, the cochlear microphonics originate.

The flagelli represent the resonators and, therefore, are elements of *transition* between the two systems. The rigid humoral system of conduction continues in the flagelli, which are elastic elements; so that any pressure exercised on the first is automatically transmitted to the flagelli.

Nature, making use of a liquid system, therefore incompressible, in order to propagate the sound pressures are aimed at addressing the stimulation exclusively towards those elements that have the function of receiving them.

We presume the existence of other elastic devices in order to explain the possibility of the intralabyrinthine propagation of the sound pressures would frustrate Nature's aim.

The problem of the round window, that should give elas-

ticity to the humoral system, would not exist in the normal function; the round window acts as a safety-device, when sounds are loud and, above all, when the pressures have no acoustic frequence.

I am relating brief and partial notices on my hearing theory, because this modality of endolymphatic circulation strengthens the hypotheses I advanced regarding the stimulation of the hair cells, while the modality of this physiological event supports that of the suggested endolymphatic circulation.

In my opinion, all the clear epithelial cells of the cochlear duct, except the hair cells and those lining Reissner's membrane, have the function of transferring nutrient fluid; they may be considered, therefore, as elements of a *sui generis* interstitial tissue, greatly differentiated, comprehended between the capillaries of the structures yielding nutrient substances and the capillaries of the structures absorbing waste products. The fluid existing in the large and small endolymphatic spaces of the cochlear duct, would assume the value of interstitial liquid.

The various hypotheses about the origin and the absorption of perilymph are well known; it is supposed to have no place of its own, so that many authors claim that it is derived from the cerebrospinal liquid; others by osmosis from the endolymph and from the vessels; according to others, both sources provide perilymph. Some of these hypotheses are denied by Lempert's recent experiments, supporting the impermeability of the walls of the membranous labyrinth.

Recently Mygind has suggested, as a source, a zone of the spiral ligament, corresponding to the insertion of Reissner's membrane, and which he called *soprastria*. In this area, in rabbits, is found a spiral vein, from which originate collaterals that run over the anterior face of Reissner's membrane, and proceed to the vasculo-epithelial zone.

I believe the cochlear source of perilymph is in this latter location; it would secrete a sufficient quantity, without resorting to other sources.

The absorption of perilymph would take place readily in the perilymphatic spaces, especially in the scala tympani.

SUMMARY.

The interpretation of some histological structures, of their vascularization and the relationship existing between tectorial membrane and organ of Corti described by the author, allows him to infer as follows: the stria vascularis secretes crystalloids, while the villi of the spiral prominence secrete plasma; that, by means of the spiral canalicular system and the cells of the outer spiral sulcus, reaches the cochlear duct, where it unites with the crystalloids, to form the endolymph.

The spiral canalicular system acts also as a safety-device, since it absorbs the amount of plasma exceeding the contingent need of the hair cells. The endolymph performing the function of transmitting the sound pressures, is absorbed in the anterior face of the limbus, while the endolymph with nutrient function circulates in the organ of Corti in a particular way and is absorbed in the limbus, by means of the interdental furrows of its outer face. Perilymph would originate from the vasculo-epithelial zone of the limbus.

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AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY.

The Annual Spring Meeting of the American Society of Facial Plastic Surgery will be held March 10-11, 1958, at Columbus, Ohio. For further details write Dr. Samuel M. Bloom, Secretary, 123 East 83rd Street, New York (28), N. Y.

USE OF GAMMA GLOBULIN IN THE CHRONICALLY ILL CHILD.

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Odessa Tex.,
and
H. L. DOBSON, M.D.,
Houston, Tex.

It is one of the unfortunate aspects of clinical practice to be repeatedly confronted with children, varying in age from 18 months to ten years, who are almost constantly ill. They are brought to the physician with acute otitis media, nasopharyngitis, tonsillitis, sinusitis, pneumonitis, and asthmatic bronchitis. Other diseases either co-exist or occur, but the respiratory tract seems to absorb the main onslaught of infection.[1] When examined during a comparatively asymptomatic interval, cervical adenitis, purulent rhinitis (chronic ethmoiditis) and serous otitis media, is the rule. Hearing loss may be alarming, and frequently is the reason for consultation; or it may be the chronic cough, or the repeated bouts of unexplained fever.

Many of these children have had, and are receiving, excellent medical care at the time of consultation. They have run the gauntlet of diagnostic and therapeutic armamentarium, leaving the child antagonistic and the parents bewildered and disillusioned. These children have received antibiotics by the gross and vitamins by the carload. They have been skin tested and desensitized. They have had cultures made followed by autogenous or stock vaccines. They have been shuttled back and forth across the country in an effort to find the "suitable climate". They have had tonsillectomies, repeated adenoidectomies, nasopharyngeal irradiation, etc., and still they become acutely ill every three to four weeks.

These procedures are not being criticized. Many such chil-

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication June 15, 1957.

dren are allergic; the value of adequate handling of adenoid tissue to preserve hearing is unquestioned, and respiratory vaccines can produce amazingly good results in the child capable of forming immunity. It is not until the entire history is unfolded that the true nature of the disability is apparent. The parent usually sums it up with the initial complaint, "He just doesn't seem to have any resistance to infection," to which the physician will probably add in his record—"poor protoplasm."

In 1952 Gamma globulin was used in a field test in Texas, Iowa and Nebraska as a prophylactic agent for poliomyelitis, and was found to be protective against that disease within certain limits.[2] Over 27,000 children, between the ages of one and 11 years of age were inoculated. Naturally, in so large a series some of the above described children were included. The result, as far as prevention of respiratory infections in this particular group is concerned, was remarkable according to parental report, with the children spending the first infection free months in years.

In 1953, Janeway[3] described a group of children with unusual susceptibility to respiratory disease with little or no discernible blood levels of Gamma globulin. These multiple recurring septic bacterial infections were in direct contrast to their normal response towards the common virus infections of childhood.

In 1954, Harris[4] reported five cases of infants and small children in which infections, both respiratory and gastrointestinal, leading to near moribund conditions failed to respond to antibiotics, but responded dramatically to Gamma globulin. Symptoms of a purulent ethmoiditis either accompanied or heralded the onset of the pulmonary or gastrointestinal infection in each case.

Originally the phenomenon of hypogammaglobulinemia was felt to be limited to children, possibly because of the failure of these cases to survive to adulthood. Recently, however, cases have been reported concerning adults[1,5]; one involved a severe surgical wound infection, the other was a patient who, over a period of years, had multiple respiratory, gastro-

intestinal and meningeal infections. Both were characterized by failure to respond to adequate, even tremendous, antibiotic dosage, only to respond immediately to Gamma globulin.

Stimulated by the works of Janeway and Harris, and with the clinical observation during the original field try of immune globulin, the first of 12 children, in whom repeated bacterial treatment-resistant infections were the outstanding clinical problem, was started on immune globulin injections in 1954. Since that time an additional 11 patients have been studied. The minimal period of observation has been 16 months, with an original nucleus having been followed for nearly three years. Below the original 12 cases are briefly summarized.

Case 1. N.M.J.—Seven-year-old female. Repeated tonsillitis and ethmoiditis accompanied by cystitis.; T & A April 15, 1954. Reccurrent attacks of acute ethmoiditis continued, accompanied by otitis and bronchitis. All attacks responded to antibiotics, but recurred every two or three weeks. Bacterial vaccine given for four months with no noticeable improvement; no history of allergy or edema; skin test negative; serum Gamma globulin 15.6 per cent; Schick test negative. Started on immune globulin October, 1954; has been followed for 30 months, during which time she has had one attack of sinusitis, which subsided spontaneously without antibiotics.

Case 2. M.F.—17-year-old female. Still's disease, severe, with ankylosis; history of repeated acute attacks of sinusitis and bronchitis throughout life; pneumonia once each winter for four years, always preceded by sinusitis; no history of allergy or edema; observed for one year, during which time she had eight attacks of sinusitis accompanied by bronchitis or laryngo-tracheitis; therapy complicated by multiple drug sensitivities; serum Gamma globulin 17 per cent; Schick test negative. Started on immune globulin January, 1955; followed for 28 months without infection, until attempt at discontinuance of injection during summer months, at which time, six weeks after the last injection she developed a typical attack. A routine dosage of immune globulin was given and the infection subsided without antibiotic.

Case 3. T.C.—Five-year-old male. Birth injury with paralysis VII and XII nerves; marked perceptive deafness; generalized poor protoplasm with suppuration of hematomas and failure of skin abrasions to heal; history of repeated upper respiratory infections accompanied by suppurative otitis medias throughout winter months; no history of allergy; periodic edema of eyes and hands, usually accompanying infection. G.U. studies negative; serum Gamma globulin 13.2 per cent; Schick test negative. Started on immune globulin February, 1955; followed for 26 months without infection; tendency to hematoma formation following injury has diminished and there has been no suppuration; edema has not been noticed since onset of therapy.

Case 4. J.N.F.—Three-year-old male. Failure of full development of right zygomatic arch and maler eminence, otherwise normal growth and development; history of acute upper respiratory infections, accompanied by otitis every two to three weeks since birth, confirmed by referring pediatrician; seen during two typical attacks consisting of

nasopharyngitis and otitis media with effusion, 22 days apart; no history of allergy or edema; skin tests negative; previous use of an autogenous vaccine from an ear culture had been unsuccessful; serum Gamma globulin 12.3 per cent; Schick test negative. Started on immune globulin December, 1954; followed for 27 months, during which time he had two attacks of rhinitis, one of which was treated with antibiotics by another physician.

Case 5. J.P.—Four-year-old female. Recurrent tonsillitis and otitis since birth; T & A age two years, following which she was free of infection for six months, then began to develop recurrent otitis. For three months prior to her first injection she had constant otitis and otorrhea, which had proved resistant to penicillin, streptomycin, triple sulfas, and aureomycin. When seen in February, 1955, she was acutely ill with purulent sinusitis, otitis media and pneumonitis. There was no history of allergy or edema; serum Gamma globulin was 14 per cent; subsequent Schick test was negative. She was started on Chlormycetin and given her initial injection of immune globulin. She responded rapidly, and has had only one attack of otitis during the 24 months that she has been followed. This subsided spontaneously.

Case 6. L.J.—Eight-year-old male. Hearing problem; history, repeated attacks of otitis all of life; tonsillectomy, two adenoidectomies and nasopharyngeal irradiation by age six; hay fever, but no edema. On desensitization regime allergic rhinitis was controlled, but repeated attacks of purulent ethmoiditis continued to occur as observed over a period of two years; with each attack effusive otitis media would occur requiring myringotomy. In the intervals the drums would remain retracted, and the hearing would be down 35-50 db.; serum Gamma globulin 10.1 per cent; Schick test negative. Started on immune globulin in March, 1955; has had no infections since. The tympanic membranes are normal, and audiometric testing shows normal hearing, except for a 20-db. perceptive loss at 4,000 cycles.

Case 7. T.W.—Nine-year-old male. Repeated attacks of otitis media, associated with respiratory infections, asthma and bronchitis; allergy both by history and by skin testing; no edema; previous attempts at desensitization failed to control symptoms; 14 attacks of purulent ethmoiditis in three years, severe enough to warrant treatment; asthma and otitis always secondary to this. Allergic rhinitis during rare infection free intervals was minimal. Serum Gamma globulin March, 1955, was 11.4 per cent; only one attack of otitis since, which subsided spontaneously. The chronic purulent ethmoiditis has completely cleared; there have been no further attacks of asthma.

Case 8. L.S.—Seven-year-old female. Hearing problem; tonsillectomy, two adenoidectomies and nasopharyngeal irradiation by age six; attacks of acute otitis media continued to occur. In the intervals a serous otitis persisted which defied treatment; hearing loss remained between 30-40 db. There was no associated infection of the sinuses, autogenous vaccine was unsuccessful; no allergy by history or skin testing; no history of edema; serum Gamma globulin 10 per cent; negative Schick test. Patient started on immune globulin in February, 1955; has had one attack of mild otitis, which subsided without medication; tympanic membranes and audiometric testing have remained normal since August, 1955.

Case 9. C.E.H.—Five-year-old male. Past history of repeated attacks of tonsillitis, rhinitis, otitis and pneumonitis with poor response to antibiotics; no improvement following T & A; no history of allergy; no edema present with infections; serum Gamma globulin less than 1 per cent; Schick test not done. Started on immune globulin February, 1955; followed for 24 months without infection.

Case 10. O.B.—Nine-year-old male. Asthma, always associated with upper respiratory infections every two to three weeks throughout winter months. Attacks of tonsillitis, otitis, rhinitis, and one episode of pneumonia; definite allergy to foods; no edema; no improvement on elimination diet; stock respiratory vaccine for four months without benefit; serum Gamma globulin 10.2 per cent; Schick test not done. Started immune globulin December, 1954; followed 26 months without infection. Two mild attacks of asthma following ingestion of chocolate which required no treatment.

Case 11. P.H.—Four-and-one-half-year-old female. Recurrent tonsillitis, otitis and sinusitis; multiple attacks of unexplained fevers with cervical adenitis; first attack of pyelitis December, 1953, was resistant to all therapy; no allergic history; no edema; serum Gamma globulin 13.5 per cent. First started on immune globulin April, 1954; received one injection a month for three months, with no infection in this interval. Injections discontinued in June, 1954, as parents felt child was cured. In July, 1954, pyelitis recurred with an upper respiratory infection. From July to November, 1954, the child had constant pyuria and repeated rhinitis and pharyngitis. Three urological consultations and a T & A produced no change in condition. In November, 1954, immune globulin was again instituted. The patient has been followed for 29 months without reinfection; all interval urinalyses have been normal.

Case 12. D.M.—Three-year-old male. Eczema began at six months of age, followed by furuncles, impetigo, chronic purulent rhinitis; pneumonia on two occasions; angina associated with agranulocytosis; repeated otitis; cellulitis of finger; suppurative axillary lymph node, followed by brain abscess, with organism sensitive to Bacitracin only; no definite allergy; eczema always associated with infection; periorbital edema with each upper respiratory infection. Serum Gamma globulin 5.1 per cent. Child had never been well enough to get D.P.T. Immune globulin was instituted in November, 1954, at the time of the brain abscess, and given in amounts of 4 cc. at weekly intervals until complete recovery. Attempts were made to drop the dosage to 2 cc., given at monthly intervals, but a three-week schedule was eventually started because of the occurrences of otitis and purulent rhinitis during the fourth week. The child has been followed for 26 months without the necessity of resorting to antibiotics; any respiratory infections that develop subside rapidly once immune globulin is given.

DISCUSSION.

Since Bruton's[6] original case in 1952, there has been an increasing interest in the problem of Agammaglobulinemia and Hypogammaglobulinemia. The Council on Pharmacy and Chemistry has recently approved[7] the use of poliomyelitis immune globulin for the treatment of these conditions. Mazzitello[8] reports on 49 cases of Agammaglobulinemia, which have appeared in the literature since 1952. Twelve articles concerning immune mechanisms and Gamma globulin were published in the year 1956. The fundamental research associated with the cases of true Agammaglobulinemia has produced an impetus in the entire immunologic field, which

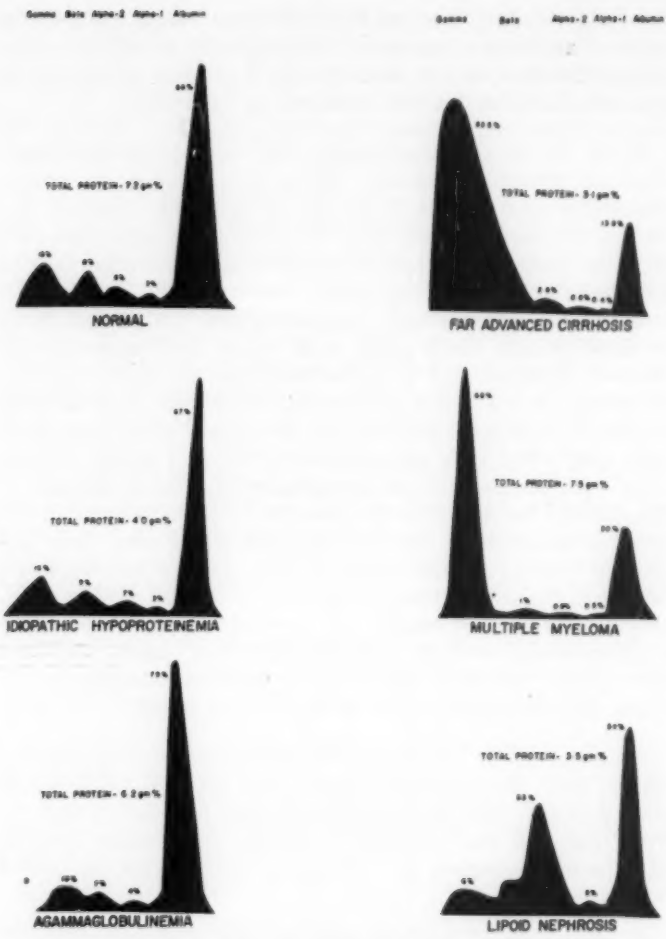

Fig. 1. Typical serum protein electrophoretic patterns.

promises to develop new concepts in the treatment of not only infectious diseases, but allergy as well.

Before the advent of antibiotics a great deal of attention was directed towards the protective mechanisms that helped

the body ward off and subdue infections. With the development of antibiotics this phase was neglected to a large extent despite the fact that if natural body immunity is lacking, no amount of antibiotics will eradicate an infection.

There are several mechanisms that have to do with resistance to infectious disease. These may roughly be divided into three groups: the first are intra-cellular factors; the second, properdin,[22] has only recently been described, and third are humeral or blood stream mechanisms (these may be intra-cellular in origin but work predominately in the extracellular and intra-vascular compartments. It is the third, or blood stream mechanism, with which this paper is concerned. It is not widely appreciated that the types and distributions of the serum protein fractions play a great part in defense against infection, and that a study of these fractions may yield very useful information in treating the patient with an unusual or complicated infectious disease.[9,10] The protein components are albumin and globulin, with the globulin being divided into Alpha one, Alpha two, Beta and Gamma fractions. Of these, all but Gamma globulins are formed in the liver. Gamma globulin is formed in the reticuloendothelial system outside the liver, either in plasma cells or lymphocytes, or both. Although albumin, Alpha and Beta globulin are related in some way to bacterial resistance, it is in the Gamma fraction that antibodies are found.

Adult values of blood proteins are reached at about one year of age. Total globulin levels vary between 1.5 and 3.06 grms. per ml., with the Gamma globulin between .7 and 1.3 grms. per 100 ml. Wolfson[11] reports an average Gamma globulin concentration of 1.20 grms. per 100 ml. in pooled human serum.

Gamma globulin determinations in the reported cases were done at the Baylor University College of Medicine, using serum paper electrophoresis, in which concentrations are reported as percentages of total serum protein present. The reasons for reporting in this manner are summarized by Jencks.[23] The average normal value for Gamma globulin using this method, as determined by testing healthy young

adults, was 15 per cent with a range of 9 per cent to 20 per cent. Using the same method all patients on a large medical service were tested. This included not only patients with infectious diseases, but cardiacs, arthritics, diabetics, etc. The average Gamma value in these patients subjected to stress was 22 per cent.

It has long been known that a person with a chronic debilitating illness usually is more susceptible to infection than a normal person. Extensive studies utilizing serum electrophoresis have shown that in such patients the serum protein pattern shows a steady decrease in albumin and a steady increase in Gamma globulin.[10] This would seem paradoxical if it is assumed that Gamma globulin's only function is the production of antibodies, and that an elevated level for the former implies the same for the latter. In such states the globulin present is either very low in specific antibody titre, an *irregular disturbance*, or is *ineffectual*, perhaps due in part to the lowered albumin. Favour[12] has advanced the theory of the immunologic blockade to explain the irregular disturbance form of Gamma globulin. In such cases it is hypothesized that bacterial infection early in life, of a relatively severe nature before the immunologic system is fully developed, may result in a paralysis to the specific antigen, which may be permanent throughout life, or continued by repeated exposure to the antigen.

Ineffectual Gamma globulin is well illustrated by cirrhosis of the liver, with extremes in low albumin and high Gamma globulin, yet marked susceptibility to infection. A similar condition exists in multiple myeloma (see Fig. 1 graphs of electrophoretic patterns).

In contrast to these are the *Functional* Hypogammaglobulinemias of nephrosis, protein starvation, and frequently lymphomas. Bacterial infections produce problems of paramount importance in these diseases, and their existence should always be considered in the diagnosis of the chronically ill child.

The final group is Bruton's congenital Agammaglobulinemia. Besides the extremely low Gamma globulin levels,

certain physical findings are said to exist. A true case supposedly is a male with an hereditary tendency to the disease. Blood isohemagglutinins are absent, and successful dermal homotransplants can be carried out. In contradiction to the acquired specific immunologic blockade, true Agammaglobulinemia represents a generalized immunologic paralysis.[13] In the cases presented in this paper, three of their histories and Gamma globulin levels might be said to conform to the classic description of Agammaglobulinemia. The majority fall in the irregular or immunologic blockade classification. There were no cases of ineffectual Gamma globulin nor functional hypoproteinemias.

Various reporters have ascribed certain physical signs or traits, which they feel are characteristic to Hypogammaglobulinemia. A breakdown of the cases showed the following to be present:

Edema[14] was present with infection in two cases; congenital malformations, not counting two cases of Mongolism were present in three cases.

Allergy, which at first one would expect to be a common complicating factor in such a group, was present in four cases. Two required specific allergic therapy in addition to Gamma globulin in order to control the main presenting symptoms. Two cases of infectious asthma responded excellently. Bowen[15] has reported a much larger series, with equally good results.

The Schick test was given to all patients in this series who had had diphtheria toxoid within the last three years as a rough estimate of their ability to form antibodies to a specific immunizing agent.[16] All cases so tested were Schick negative.

The mechanism of treatment was extremely simple. Poliomyelitis immune globulin, prepared from human plasma collected by the Red Cross, was first used. Commercial immune globulin, extracted from serum of professional donors is now available. Because of the known greater effectiveness of convalescent serum,[17] and the disproportionately high incidence of staphylococcus in the North and East, an attempt

was made to use globulin prepared from donors in the same general area as the test cases.

The amount administered was from 2 to 4 cc.[18] each month, which is well within the half life of six weeks[19] considered to be the limit of effectiveness of the immune globulin.

Antibiotics were withheld and, except in rare instances, were not necessary. It was originally hoped that some superimposition[20] of active immunity would occur on having the patient repeatedly exposed to infections in a modified form. Clinically this failed to develop with a severe respiratory infection following within four to six weeks any attempt to discontinue the immune globulin. Such findings, although disappointing, would coincide with the theory of immunologic blockade.

It is possible that in the future some more effective subfraction[21] of Gamma globulin, derived from convalescent serum with a measurable specific bacteria antibody titre, will be available and provide a much greater degree of protection.

SUMMARY.

Twelve cases of chronically ill children, in whom repeated infections by respiratory pathogens failed to respond to the usual methods of treatment, are reported.

Serum Gamma globulin levels, as well as some of the clinical aspects of these cases, are compared with the newly evolved classifications of globulin disturbances.

In each of the reported cases the response to repeated injections of immune globulin was excellent.

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MALIGNANT TUMORS OF THE NASO-PHARYNX.*

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The incidence of malignancies of the naso-pharynx is known to form a very small group of all malignant tumors of the body. The lifetime series of any individual otolaryngologist is, therefore, very small. The frequency of the disease is estimated generally at about 0.3 per cent of all body malignancies. An exception to this is that among the Chinese people, this appears to be the most common tumor, with figures as high as 25 per cent being reported in the literature.

This series consists of the report of 87 cases of naso-pharyngeal carcinoma seen during the period 1940-1955, with a review of five personal cases. I have included no cases under 17 years of age in this review.

Pathology. Table No. 1 shows the type of lesion present in this series. There is always present among the pathologists a wide degree of controversy regarding the nomenclature used to differentiate the tumors that are present in this group. This is especially true in regard to the transitional cell carcinoma, the lympho epithelioma and the anaplastic carcinoma, along with the basal cell carcinoma. There is a large group of cases under the general heading "carcinoma" for this very reason.

The average age of all the patients in the study was 53 years. It is considerably more common in men than women, at the ratio of 7 to 1. In the group of lympho epitheliomas, of the 29 cases reviewed, there was only one female and seven Chinese males which constituted 24 per cent of all this type of tumor. In the complete review of 87 cases, there were 16 of Chinese nationality. This is a very pertinent fact when it is realized that there is a very small Chinese population in Metropolitan Toronto and the Province of Ontario.

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*Read at the meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, Inc., Toronto, Canada, Jan. 31, 1957.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication September 27, 1957.

Symptomatology. The importance of the symptomatology can not be overstressed, as this is often the clue to earlier diagnosis. McWhirter, reviewing over 100 cases, noted that there was an average delay of 9.5 months from the onset of symptoms to the institution of treatment. The patient delay was 1.7 months, the rest of the delay being due entirely to the medical profession. In this series of cases, the average delay was 7.9 months; the medical delay was approximately three months. This is still sufficient delay to jeopardize the im-

TABLE I.

TYPES OF MALIGNANT LESIONS OF NASOPHARYNX

1940 - 1955

BASAL CELL CARCINOMA	3	ADENOCARCINOMA	1
SQUAMOUS CELL CARCINOMA	22	CARCINOMA	18
LYMPHO-EPITHELIOMA	29	RETICULUM CELL SARCOMA	4
TRANSITIONAL CELL CARCINOMA	4	ANAPLASTIC CARCINOMA	6

TOTAL CASES 87

provement in a five-year survival rate. In Godtfredsen's (1944) extensive review of the symptomatology of malignant naso-pharyngeal tumors of 454 cases, he grouped the symptoms under four headings: neuro-ophthalmological, glandular, naso-pharyngeal and otological. We have chosen the same grouping to classify the initial symptom and the symptoms in the established disease.

Table No. 2 shows that neuro-ophthalmological symptoms were initially present in 15.8 per cent of this series. The most common symptom was pain over the distribution of the

first and second divisions of the Vth cranial nerve. The most common ophthalmological finding was the paralysis of the VIth cranial nerve. Godtfredsen felt that these signs were found in the early stages of the disease as well as the late stages, contrary to previous views held. This has been true in this group.

The glandular or metastatic symptom constituted 43.4 per cent of the initial symptom. It is perfectly obvious that in this development there is a widespread dissemination of the

TABLE II.

PRESENTING FEATURES IN MALIGNANT DISEASE OF THE NASOPHARYNX

	TORONTO		GODTFREDSEN	
	INITIAL SYMPTOM	SYMPTOMS INESTABLISHED DISEASE	INITIAL SYMPTOM	SYMPTOMS INESTABLISHED DISEASE
NEURO-OPHTHALMOLOGICAL	15.8%	33.6%	33.3%	38.8%
GLANDULAR	43.4%	68.9%	26.1%	70 %
NASOPHARYNGEAL	24.1%	44.4%	24.1%	40%
OTOLOGICAL	17.7%	31.1%	15.4%	49%

primary disease. The usual involvement is the deep cervical group of glands which lie along the anterior border of the sternomastoid muscle. Occasionally glands are found in the posterior triangle of the neck and the region of the anterior border of the trapezius. McWhirter points out that it is unusual to find this distribution of glandular involvement in any other disease.

Naso-pharyngeal symptom was noted in 24.1 per cent of cases. This consisted mainly of nasal obstruction, nasal hemorrhage, or increased naso-pharyngeal discharge.

Otological symptoms were primary symptoms in 17.7 per cent of cases. This was usually due to the spread of the disease within the naso-pharynx, causing obstruction of the Eustachian tube, thereby producing tinnitus and loss of hearing. Table No. 2 also shows the comparison in the presenting

features in malignant disease of the naso-pharynx of the Toronto series with the Scandinavian series of Godtfredsen. There is a significant difference in the primary symptom of the metastatic group, this being suggestive of a poorer outlook with reference to five-year survival rate.

It is not difficult to realize, in studying these groups, that specialists in different fields are frequently consulted; the otolaryngologist often being the last to be consulted. Trotter in 1911, and Gorden New in 1922, were pioneers in establishing the symptom complex of carcinoma of the naso-pharynx. Trotter described the anatomical cause of the symptoms for the first time. New, in his paper, reported that 74 of 79 cases, were operated upon for varying reasons not related to the naso-pharynx. There has been a definite step forward in this respect, and this occurred only in six of the present series. In the symptoms of the established disease, as noted in the two series, there is very little difference except as regards the otological symptom. This may possibly be explained on the grounds that the present series deals entirely with carcinoma, and the sarcomatous group tends to be more bulky and thus to lead to obstruction of the Eustachian tube orifice. The disease tends to follow a definite pattern from its primary site in its extension to the surrounding tissues, at the base of the skull cervical lymph nodes, and is then disseminated throughout the body.

Method of examination of the naso-pharynx is certainly very important and it is well known to every otolaryngologist. I do feel that it is worthy to recall a few of the important points, difficulties and pitfalls of examination of the naso-pharynx. Post-nasal mirror is probably one of the best methods of examination of the naso-pharynx, but it is often difficult, even after applying local anesthetic, to get a satisfactory view. Repeated examinations are often necessary and not always successful. The use of the naso-pharyngoscope can give a limited view of the vault and lateral wall of the naso-pharynx with special emphasis on the Eustachian tube orifice. There is always a blind spot in relation to the most important area of the naso-pharynx, Rosenmuller's fossa, this being due to the placing of the bulb distal to the mirror in

these instruments. I have recently used the lighted Van Treeck speculum, which is introduced through the mouth and enables a very satisfactory examination of the naso-pharynx to be carried out. The only problem is to obtain this instrument, as it is made in the eastern zone of Berlin.

In some patients it is possible to carry out a satisfactory direct examination of the naso-pharynx after previous preparation with local anesthetic, using a Yankauer speculum. Digital examination can also be carried out at the same time.

TABLE III.

CARCINOMA OF NASOPHARYNX

1940 - 1955

SURVIVAL	NO. OF CASES	ALIVE	% SURVIVAL
1 YEAR	87	48	55.2%
3 YEARS	80	18	22.5%
5 YEARS	77	7	9.1%

I have found this method exceedingly difficult, as even the most co-operative patients find this procedure intolerable. In my very limited experience, I found it much more satisfactory to carry out this type of examination under general anesthesia. A more thorough, relaxed examination is possible. I am also able to use my index finger to direct the forceps to the suspicious area and obtain a satisfactory biopsy in small sessile types of lesions. X-rays of the naso-pharynx should always be taken to demonstrate the soft tissue outline of the naso-pharynx. This will often show a lesion arising from the posterior wall of this area. X-rays of the

base of the skull should also be taken routinely. These will often demonstrate a lesion arising from the lateral wall of the naso-pharynx and involvement of the base of the skull.

The treatment of these cases, due to the inaccessibility of the area to surgery, has been solely irradiation. Treatment carried out at the Dunlap Institute is to supply about two-thirds of the cancer lethal dose by external radiation at 400 kv. and using a four or six portal technique. The remaining one-

TABLE IV.

NASOPHARYNX 5 YEAR SURVIVAL RATES	
SIMMONS AND ARIEL	9.8%
NEILSEN, J.	24%
NEW, G.B.	13%
FURSTENBURG	2.5% (2 YEARS)
KASABACK, H.H.	16.6%

third is delivered by local application of radium in the form of a post-nasal plug. The above treatment plan includes tumor dosages of roughly 3,500 to 4,000 *r* units delivered by external radiation in three-and-a-half to four weeks, and supplemented immediately by a radium dosage of 2,000 *r* at a depth of 0.5 c. on the surface of the applicator which is used. In the majority of cases, application of external radiation with 400 kv. alone was used. With the widespread extension of the disease, it was felt that the local application of radium was not indicated in many cases.

Table No. 3 shows the number of cases that have survived this treatment at one-year, three-year and five-year survivals. It will be noted in Table No. 1 that 57 cases already had

metastatic involvement in the cervical lymph nodes. The five-year survival rate is remarkably poor, but compares favorably with figures from other institutes. Since 1954, Cobalt 60 has been available for the treatment of these tumors. At first the only cases were those untreatable by other means, because of the extent of the disease or previous radiation. They were treated only for palliation. It is now the policy to treat all cases of naso-pharyngeal tumors with Cobalt 60. Of the cases treated, solely and radically by this means, in a series of four, three are alive and well, one over two years and two over one year. The fourth is alive but receiving treatment for metastases. This treatment in all cases has been supported by a radium post-nasal plug. Allt thinks that even though the period is a little short to prognosticate as to the five and ten-year survival rates, early indications are definitely encouraging, and one would look forward to ten years to fulfill the present hopes of a better overall result.

Following are five personal cases seen in the past two-and-a-half years:

Case 1. Mr. J.D., age 73, white male, admitted to hospital on April 1, 1955, complaining of the loss of hearing for three months and diplopia for two months. The positive clinical findings were a left VIth and a left Vth cranial nerve paralysis. Careful naso-pharyngeal examination failed to reveal any evidence of tumor. His admitting diagnosis was a naso-pharyngeal tumor. X-rays were taken of the naso-pharynx and of the base of the skull and showed no evidence of tumor. Audiometric studies revealed bilateral presbycusis. Vestibular function tests were carried out, and all tests were within normal limits. Neurosurgical consultation added little to the picture, except that when lumbar puncture was carried out pressure was found to be within normal limits, but the proteins were raised to 64 mgs. per cent. Diagnosis of possible basilar artery aneurysm was made. A biopsy was taken of the left Rosenmuller's fossa, and the pathological report was chronic inflammation. It was decided to discharge the patient from hospital, and he was to be carefully followed up in the out-patient clinic. The patient returned again on June 15, 1955, and it was noted that there was a fullness in the left Rosenmuller's fossa. He was re-admitted to hospital and under general anesthetic a biopsy was obtained from the naso-pharynx. This time an adenoid curette was used, and a small portion of hard bone was removed in the region of the left sphenoid. Pathologist reported this to be squamous cell carcinoma. The patient was placed on a course of high voltage, 400 kv. for four weeks, but his condition rapidly deteriorated, and on September 16, 1955, the patient died in hospital. Postmortem was carried out. A large mass was found in the naso-pharynx, extending through the anterior wall of the left sphenoid and eroding the posterior lateral wall of the posterior ethmoids on the left side, extending into the left orbit. The Vth and VIth nerves were surrounded by tumor mass, and there was a direct invasion of the cavernous sinus with squamous cell carcinoma.

Comment: From the beginning this patient was suspected to have a naso-pharyngeal carcinoma, but the primary tumor appears to have extended to the base of the skull rather than directly into the naso-pharynx. The increased protein in the C.S.F. was suggestive of involvement of the meninges.

Case 2. Mrs. I.K., 47-year-old white female, who was admitted to the neurosurgical unit on May 28, 1955, complaining of pain and numbness over the left side of the face for six months, and sudden onset of diplopia three weeks before admission. A previous history showed that the patient had been treated for syphilis in 1947. Positive clinical findings were reduced sensation over the distribution of the first and second divisions of the trigeminal nerve on the left side. There was a proptosis of the left eye downwards and laterally, with restriction of eye movements in a medial and upward direction. X-rays of the naso-pharynx showed a mass in the post-nasal space with extension to the left sphenoid and the posterior ethmoidal cells, and erosion of the orbital plate. The patient was then referred to the ear, nose, and throat service, and examination revealed the presence of a large mass in the left lateral wall of the naso-pharynx. Under general anesthesia palpation was carried out, and this was found to demonstrate the origin of the tumor in the left Rosenmuller's fossa, extending forward above the left Eustachian tube orifice. Biopsy was taken from this site and reported as an undifferentiated sarcoma. The patient was given a course of Cobalt 60 during a four-week period. This was followed by a complete melting of the tumor mass, the return of the eye to normal, and the patient has been asymptomatic since that time.

Case 3. Mr. J.S., 60-year-old white male. In July, 1954, visiting Canada for the wedding of an immigrant daughter, it was noticed that his hearing appeared to be markedly reduced. The patient was referred for treatment of deafness. On examination, it was found that he had a bilateral serous otitis media. On examination of the naso-pharynx, a large tumor mass was found to arise from the roof. Under local anesthetic a large piece of mass was removed and the pathological report showed reticulum cell sarcoma. The patient returned home. One week later he was given a course of high voltage 400 kv. He is now alive and well after three-and-a-half years.

Case 4. Mr. S.W.W., 40-year-old Chinese male, seen in July, 1955, with a history of a lump in the left side of the neck, a portion of which had been removed three months previously by his general practitioner and sent for pathological examination. The report at that time was lympho-epithelioma. The patient refused to have any further treatment, but returned on the advice of friends. Examination of the post-nasal space showed a mass in the naso-pharynx. The patient was put on a course of high voltage 400 kv. but did not attend regularly. In October, 1955, he developed multiple secondaries in the ribs, vertibrae and hip. He died on January 29, 1956. No postmortem was obtainable.

It has been noticeable among the Chinese patients in the groups reviewed above that there is a longer delay among these people in presenting themselves after the initial symptom is present. It is also hard on account of the language difficulty to convince them of the need for regular attendance for treatment.

Case 5. Miss M.W., 60-year-old white female, seen in August, 1956, who had been complaining of nasal-obstruction for three years. She was treated with nose drops by various medical practitioners. Her condition became markedly acute one year ago. She also had a three-week history of left-sided headaches. On clinical examination it was found that there was a large mass in the left lateral wall of the naso-pharynx extending on to the vault. Palpable glands were present in both anterior triangles of the neck. Under general anesthesia careful palpation of the tumor mass was carried out and a biopsy obtained. The pathological report was anaplastic carcinoma. This patient was put on a course of Cobalt 60 and teleradium. There was a rapid shrinking of the tumor mass with disappearance of the glands in the neck. The patient was seen at frequent intervals, and on January 13 it was noted that there was a small area of thickening behind the left Eustachian tube. The patient was readmitted to hospital, and under general anesthesia palpation of the post-nasal space was carried out. It was noted that there was an indurated area situated behind the left Eustachian tube, extending for a short distance on the posterior wall of the naso-pharynx by direct continuity. A biopsy was taken from this site, and a report of anaplastic carcinoma was obtained. Three days later, a post-nasal plug, containing 40 mgs. of radium, was inserted into the naso-pharynx under general anesthesia. This was left in position for 50 hours. It is too soon to give any prognosis in this case.

These five cases illustrate the different presenting symptoms which have been my personal experience in the last two-and-a-half years. It demonstrates some of the problems that can be encountered.

Summary: the presentation of the review of 87 cases of malignant tumors of the naso-pharynx regarding pathology, symptomatology and treatment, with short case histories of five personal cases.

I thank Dr. C. Ash, Director, Dunlap Institute of Radiotherapy, for all of his kind help in preparing this paper.

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IMPORTANCE OF EARLY DIAGNOSIS OF MALIGNANCY IN THE TONSIL: CASE REPORT.

ALBERT P. SELTZER, M.D.,*

Philadelphia, Pa.

Malignant growth of the tonsil is frequently overlooked, since its early manifestation so closely resembles an inflammatory process, with swelling and pain in the tissues of the gland and its surroundings.

A rule which should be emphasized is that if any such tonsillar condition, limited to one side of the throat, does not respond promptly to local treatment, a biopsy should be done in order to assure a definite diagnosis of the nature of the process.

The following case report is significant:

Case E. L.: A 47-year-old negro was admitted to the writer's hospital service on July 3, 1956. He had been admitted to the hospital first, in October, 1953, with the complaint of increasing frequency of attacks of sore throat accompanied by some difficulty in swallowing. A tonsillectomy was done, followed by irradiation, with some temporary improvement.

He returned to the hospital in 1954 for further treatment, because of recurrence. The lymph nodes of the left cervical chain were enlarged and hard, and were recognized as due to metastases from the primary condition in the tonsil. The diagnosis of malignancy was then recorded.

Operation was not advised because of the extent of the metastases, but irradiation was continued. There was more or less pain in the left throat, which continued to increase in severity and extent. A craniotomy was done by multiple openings in the skull with the idea of reducing intracranial pressure which might be caused by metastases to the brain with resulting pain, which had been uncontrollable.

Later, when the pain had been relieved by other measures, but had increased, a frontal lobotomy was done as a final means of counteracting the pain, which had not responded to sedatives.

On August 3, 1956, when the patient was admitted to the writer's service, there was severe bleeding from the left tonsillar region. Carotid ligation was proposed for control of the constant excessive bleeding, but this was not attempted because of the extent of the cervical metastases,

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Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Sept. 20, 1957.

and also because of the weakening of the blood vessels by repeated irradiation.

The patient was in a very serious condition from loss of blood, and emergency measures were necessary. For this reason a tracheotomy was done under local anesthesia, and the oral pharynx was firmly packed with gauze. The bleeding was considerably slowed by these measures and the pharyngeal packing was later gradually removed.

To compensate for the large amount of blood lost, a 3000 cc. blood transfusion was given; also cold fluids and sedatives. The bleeding continued at this time regardless of any measures taken, and on July 29 a maximum amount of radiation was given. By September 15, bleeding had become excessive again, so ice packs were applied, and Dumerol was given. Epinephrin was not tried as it was contraindicated by the Thorazine, which had been used. Transfusion was continued daily. By November 17, there was bleeding both from the tracheotomy opening and from the mouth; this continued during the following day, with frequent coughing. Sedatives gave no relief. Death occurred on December 18, with no abatement of the hemorrhage up to the end.

Report of the postmortem operation specified: Liver slightly enlarged (metastases not apparent), but other body organs were all negative; general, extensive metastasis to the lymph nodes of the left side of the neck. Diagnosis: Squamous cell carcinoma (primary in the tonsil).

COURSE IN RECONSTRUCTIVE NASAL SURGERY.

The Department of Otolaryngology, University of Cincinnati College of Medicine, announces a Course in Reconstructive Surgery of the Nasal Septum and External Pyramid. The course will be under the direction of Dr. Maurice H. Cottle, Professor of Otolaryngology, Chicago Medical School, and a selected faculty, in association with the American Rhinologic Society, April 12 through April 19, 1958. Class membership limited. Tuition $350.00. Please apply to Dr. Henry M. Goodyear, Professor of Otolaryngology, College of Medicine, Eden and Bethesda Avenues, Cincinnati 19, Ohio.

ABSTRACT OF THE PROCEEDINGS OF THE SEVENTY-EIGHTH ANNUAL MEETING OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION

Washington, D. C.,

May 3, 1957

LEROY A. SCHALL, M.D., President.

SAMUEL SALINGER, M.D., Abstract Editor.

SOME UNSOLVED PROBLEMS OF AVIATION OTOLARYNGOLOGY.

PAUL A. CAMPBELL, Col. U.S.A.F. (M.C.)

Among the problems which the Air Force is seeking to solve are noise production, noise sources and the media through which the energy is transmitted. Methods of cancellation of wave sources and conversion of noise into useful energy have been the subject of considerable research. Future problems have to deal with the behavior and the reactions of the human being in a gravity free field, such as envisioned when space travel becomes a reality. When the vestibular apparatus and the kinesthetic sense are inoperative through elimination of the gravity vector what will be the effect on orientation? Another problem for the future is the construction of a completely sealed cabin, in which pressurized air and oxygen have to be provided for real high altitudes, and means devised for utilizing exhaled products so as to reduce the weight of the vehicle.

Pressurization of cabins has thus far reduced the incidence

of ear discomfort, air sickness and oxygen want to a marked degree, as the statistics reveal; thus considerable progress has been made for the benefit of the increasing numbers of commercial air travellers as well as for military personnel.

DISCUSSION.

Dr. Norton Canfield pointed out that groups investigating noise problems have come up with some valuable observations and suggestions. Cancellation of noise, however, is a thorny problem which merits and is receiving a great deal of attention.

The problems of life in a gravity-free medium suggest the possibility of the human being's developing a substitute sense to compensate for the elimination of the equilibrium senses. Dr. Caufield inquired as to why cabin pressure is not increased at the moment of takeoff.

Dr. John A. Bordley remarked that standards of measurement have changed because of the enormous increases in sound, speed, etc. Also he believes we will have to know more about speech adaptations to new environments. He drew a picture of man in his new orbit leaving much of it to speculation a la Buck Rogers!

Dr. Campbell replied to Dr. Caulfield's query by stating that cabin pressures are gradually increased as the plane is taxying before takeoff.

HOMESPUN LARYNGOLOGY.

LYMAN RICHARDS, M.D.

Dr. Richards referred to the traveller returning from a long arduous journey, whose chief complaint was "sand in his shoes." The situation is the same with the laryngologist in the case of patients seeking relief from symptoms unassociated with any visible pathologic signs. He mentions the frustration which comes over the sincere doctor seeking to explain the complaint in the absence of tangible objective findings. Laryngologists are exposed to this type of patient because a visit to the doctor does not involve time consuming dressing and undressing, accessory examinations and extensive laboratory and mechanical diagnostic procedures. Yet though the otolaryngological examination fails to disclose positive findings the physician is haunted by the fear that he may have overlooked something very vital, as has occa-

sionally happened when a malignancy turns up subsequently. The multiplicity of etiologic possibilities makes it incumbent on the examiner to be thorough and painstaking, and not to give way to the impulse of prescribing his favorite remedy for nondescript complaints, such as vitamins, diets, physiotherapy, or whatever his particular hobby may be. It involves the investigation of the patient as a whole, taking into consideration his environment, personality, heredity, domestic situation, etc. The separation of psychic and somatic symptoms requires patience, insight and sympathetic understanding.

DISCUSSION.

Dr. Frederick T. Hill found himself in complete agreement with Dr. Richards in a consideration of this perplexing problem. Thoughtful physicians are always on the alert lest they fail to identify a true lesion in a patient whose symptoms seem to indicate merely a functional disorder. The percentage of functional overlay varies in different specialties, as Dr. Hill discovered on polling the various departments at his hospital. It was as low as 10 per cent in orthopedics and as high as 42 per cent in internal medicine and cardiology. He finds that it is only through careful painstaking history taking and scrutiny of every bit of evidence that can be elicited from the patient as to background and environmental factors that the true status can be established. This takes time and is inconsistent with the practice of seeing a large number of patients in a short space of time. It is the only point on which Dr. Hill took issue with the essayist, who mentioned seeing 30 to 40 patients a day.

Dr. Richards responded by stating that he had found in his tours about the country that many men had outstripped him in the number of patients seen in a restricted period of time, which was somewhat baffling. He wishes to emphasize one factor that seems to lead many men astray in obscure cases, namely, a tendency to follow a hobby such as giving vaccines, altering the diet, or applying caustics to the turbinates. There are so many angles to this problem that often confuse and lead one astray. The main point of his argument is that one should never be put into the position of having overlooked a serious lesion, and this is a matter that conscientious physicians are ever bearing in mind.

FUNDAMENTAL PHYSIOLOGICAL FACTORS TO BE CONSIDERED IN SPEECH THERAPY.

W. W. WILKERSON, JR., M.D.
and
FORREST M. HULL (By Invitation).

Analysis of human speech production involves a consideration of several physiologic systems functionally integrated. In addition to the breathing mechanism, the larynx and the resonators, a fourth factor is involved, namely, the articulators. Dr. Wilkerson showed how the latter, which are primarily concerned with more basic functions, are brought into play secondarily for the production of speech. A number of conditions are enumerated which may interfere with this function, thus affecting the production of speech. Among the conditions listed are brain damage, abnormalities of anatomical structures, lesions of various cranial nerves and central nervous system, etc. Several cases were presented to illustrate these points. Training and retraining in some of these cases may restore the normal mechanism, while in other cases compensatory movements may be developed to accomplish a greater or lesser degree of rehabilitation.

THE MECHANISM OF PHONATION.

SIR VICTOR NEGUS.

Dr. Negus presented an analysis of the mechanism of phonation, in which he refuted the theory recently advanced by some French authors to the effect that the vocal cords in phonation are activated by clonic contractures rather than being in a state of tonic contracture, as hitherto held. This new theory holds that phonation at a certain pitch is the result of an equal number of stimuli from the higher centers. Dr. Negus refutes this argument by showing first that the

fastest voluntary muscle known is in the wing of a fly which vibrates at the rate of 330 times per second. In his experience he knows of no human muscle capable of the same, and he cannot conceive of the thyro-arytenoids in this role. Besides, when one considers the higher pitches of over 2000 cycles per second, which are reached by many singing voices, the theory becomes more improbable; furthermore, it has been proven in laboratory experiments that when the recurrent laryngeal nerve is stimulated by an electric current, the action potentials coming out of the muscle will correspond to the stimuli only at low registers, such as 50 to 60 per second. When the stimuli are stepped up to 250 and higher the recorded potentials become irregular both in amplitude and distribution. If the theory of clonic contractions were to be tenable the output of potentials should correspond accurately to the rate of the stimuli.

DISCUSSION.

Dr. Louis H. Clerf confessed that he was somewhat confused by this theory, which to be clearly understood would have to be set forth in simpler terms. As for Dr. Wilkerson's presentation he finds it of practical value because of his interest in the rehabilitation of the laryngectomized patient. The art of speech therapy is being developed to a stage where it is offering considerable relief to these handicapped individuals, and gives promise of further development. He agrees with the essayist on the importance of good dentistry as a factor in the regulation of articulation. A case in point was a patient with a prominent torus palatinus, in which a denture covering the mass was less effective than one which permitted it to remain exposed.

Dr. Fred Dixon also recognized the valued service of the speech therapist in our field. The importance of good articulation and properly modulated voice cannot be overestimated when applied to individuals who are in contact with the public through various communication channels. He feels that in many cases clearer tones can be obtained by raising the register, since he believes that the lower registers frequently tend to glottic trauma.

Concerning vocal nodules, he asked Dr. Wilkerson whether he relies on surgery entirely or does he recommend vocal therapy. Also as to his advice to patients after removal of nodules in the matter of complete voice rest, use of a whisper, etc.

Dr. William J. McNally called attention to the great amount of research conducted by Dr. Negus in the laboratory and in the zoo, which commands universal respect and sets him up as an authority. He would recommend that every laryngologist keep Dr. Negus' book on the *Mechanism of the Larynx* by his bedside for careful leisurely reading.

Dr. Wilkerson closed his portion of the discussion by starting that he does not rely on surgery alone for the relief of vocal nodules. In the early stages absolute voice rest and vocal therapy later may be very effective. If surgery is resorted to, the period of vocal abstinence de-

pends on the degree of postoperative reaction and the healing process. He usually insists on voice rest for a month, and whispered voice later.

Dr. Negus closed by regretting the absence of contradictory criticisms; however, he thought that most of the members would like to give the matter some study before offering any opinion. He was grateful to Dr. McNally for his kind reference to his book, but pointed out that one physician had called his attention to the fact that it weighed six and one-half pounds, which would hardly be considered comfortable for reading in bed!

MALIGNANT LYMPHOMA OF THE PHARYNX.

STANTON A. FRIEDBERG, M.D.,
ROBERT H. EDWARDS, M.D.,
and
GEORGE M. HAAS, M.D.

Dr. Friedberg presented an analysis of six additional cases of malignant lymphoma of the pharynx which, added to the ten cases previously reported, furnished the basis of the present study. It appears that "malignant lymphoma" embraces a variety of growths, including types of lymphosarcoma as well as Hodgkins' disease. It is difficult to classify these growths on a histologic basis because there appear to be transitions from one form to another. While the tumor may be localized for a long time, systemic involvement appears to be inevitable. Prognosis seems to depend more upon the length of the remissions rather than upon the histologic picture. Early systemic involvement usually means a rapid downhill progression. Longer remission (60, 56, and 52 months in three cases) implies longer survival. Of 15 determinate cases, nine died of the disease, three are living without evidence of generalized disease although two of them are of recent date.

X-ray therapy and chemotherapy are effective in controlling the disease for varying periods of time, but apparently have no curative value.

Lesions in the pharynx such as abnormal or recurring

lymphoid masses, unilateral tonsil hypertrophy or unilaterial smooth swelling of the pharynx should always arouse suspicion of malignant lymphoma.

DISCUSSION.

Dr. Alden H. Miller found 15 cases of lymphoma of the pharynx in the files of the Los Angeles General Hospital in a ten-year period. Eleven were diagnosed as lymphosarcoma, lymphocytic and lymphoblastic malignancies, and three as reticulum cell sarcoma. One-third of the patients died within two and a half years, three were free of symptoms after five years. Nitrogen mustard relieved pain but did not halt the process. Dr. Miller emphasized the importance of obtaining a large block of tissue for histologic examination, since needle or punch biopsies do not furnish enough tissue for thorough study.

Dr. John B. Erich agreed that these various types of neoplasm must have a common origin. A review of the material in the files of the Mayo Clinic reveals a total of 171 cases of malignant lymphomas of the pharynx in which the diagnosis was lymphocytic sarcoma in 76 per cent, reticulum cell sarcoma in 19 per cent, Hodgkins' disease in 5 per cent and giant follicular lymphosarcoma in 1 per cent. They found lymphoblastoma to be very malignant and reticulum cell sarcoma very resistant to irradiation. External irradiation is effective at least in halting the process in most cases, and is superior to seeding, which was formerly employed. The prognosis, however, is bad, and it is impossible to predict the duration of the disease.

Dr. Friedberg, in closing, found it interesting that Dr. Miller had encountered almost the exact number of cases in the same period of time. While agreeing that the reticulum cell sarcomas are generally thought to be the most malignant, it was nevertheless surprising to note that some of his cases in this category survived the longest. This contrasted remarkably with other cases where the growths were more mature, and yet the survival periods much shorter. It is, therefore, quite evident, as Dr. Erich pointed out, that one cannot predict the outcome just from the histologic appearance of the tissue. The problem is still more confusing when occasionally a pathologist, viewing the same specimen at intervals, will render conflicting opinions. Another difficult matter, as mentioned by Dr. Miller, is the distinction between response to infection and hyperplasia. He has seen cases diagnosed as hyperplasia of lymphoid structures who were dead in six months from disseminated lymphoma.

Regarding different types of which Dr. Erich spoke, the author seemed to think that classification all depended upon the particular pathologist's training and his concept of the disease. Interpretation seems to vary widely in different communities. The unpredictability in these cases was well represented by a 96-year-old man with a follicular type of growth considered more benign but who succumbed more rapidly, whereas the more malignant reticulum cell growths in some cases survived longest.

THE ROLE OF RHINOLOGY IN PITUITARY SURGERY.

OSCAR HIRSCH, M.D. (By Invitation).

Dr. Hirsch gave the history of the intranasal approach to the pituitary gland, culminating in his trans-septal technique which he has employed since 1910 in approximately 350 cases. Two hundred seventy-seven patients were operated upon prior to 1938 (preantibiotic era) with a mortality of 5.4 per cent. During the antibiotic era (1944 to 1957) 75 operations were performed with only one death. Thirty-five of these are living or have survived for five years or more, 24 of them with lasting improvement.

Supra-diaphragmatic tumors are not amenable to this technique. Intrasellar growths, recognized by expansion of the sella, are ideal for the trans-septal operation.

Tumors which are both supra- and intrasellar are the most frequent type encountered, and can be operated on via the septum, although only the intrasellar portion is accessible. Since there is no other method of attacking this type of growth, the Hirsch operation offers at least some hope of relieving the optic pathways of strangulation. The post-operative use of radium has been of great help in retarding further growth.

DISCUSSION.

Dr. Shirley Baron related a case of pituitary cyst in a 53-year-old woman, in which the cyst was evacuated via Dr. Hirsch's approach, and a radiopaque solution injected which outlined it clearly. Cobalt 60 was introduced within the cavity and the patient's vision returned; however, after seven months blindness recurred in one eye. The cyst was again incised but no fluid was found. A year later the other eye became blind. Dr. Baron speculated on the possibility of the damage having resulted from over-exposure to the cobalt.

Dr. Francis L. Weille called attention to one item which Dr. Weille omitted to mention, namely, the low mortality in his series (only 1 per cent) as compared to the mortality of 30 per cent from the trans-cranial approach. In his clinic all of these cases are now seen by Dr. Hirsch, along with the endocrinology group.

Dr. Robert C. McNaught stated that they had done 15 of these cases at Stanford, and that the ophthalmologists and the Tumor Board refer them routinely to his department. They recently employed Hirsch's technique for the removal of a normal pituitary in a case where the object was to produce the effect of a complete adrenalectomy. It was accomplished despite the fact that it is more difficult when the posterior wall

of the sphenoid is normal instead of being thinned out by pressure of the growth.

Dr. Hirsch expressed his appreciation of the attention given his technique by the San Francisco group. He credits the late Dr. Lewis Morrison, who became interested after seeing Dr. Hirsch and invited him to lecture on the subject at a meeting of the Pacific Coast Society. In Boston, Dr. Schall has also been most co-operative in this respect.

Replying to Dr. Maxwell, Dr. Hirsch would like to dispel any fear of danger in this operation. With a good knowledge of the anatomy any good rhinologic surgeon should be able to accomplish it without too much training.

Dr. Hirsch stated that he would not recommend the operation for a normal gland. He believes this is more properly in the realm of neurosurgery. This also applies to the supra-diaphragmatic tumors which belong to the neurosurgeon; however, the growths that are below the diaphragm, the cases with hemianopsia and atrophy, are definitely in our field. As for Cortisone, he sees no need of it, since there is very little shock in these cases; also, the danger of hemorrhage is minimal.

MEDICAL AND SURGICAL MISADVENTURES.

FRANCIS L. LEDERER, M.D.

Dr. Lederer presented a broad analysis of the many factors which may lead to misadventure in the practice of otolaryngology. From the very first contact with the patient, the physician passes through the various stages of history taking, general appraisal of the patient, examination and therapy, each of which is fraught with pitfalls into which the unwary may fall. In addition, many errors are due to preconceived notions, partiality to certain types of therapy and individual hobbies which may or may not fit the case; furthermore, surgical risks add to the possibilities of error and misadventure.

The list of items elaborated by Dr. Lederer is formidable, and it all adds up to the physician himself, his temperament, education, patience, perspicuity, thoroughness and skill.

No discussion.

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(Secretaries of the various societies are requested to keep this information up to date).

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Meeting: Palmer House, Chicago, Ill.

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Place: Mark Hopkins Hotel, San Francisco, Calif., May 21-23, 1958.
Place: The Homstead, Hot Springs, Va., March, 1959.

SECTIONS:

Eastern—Chairman: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadephia 40, Pa.
Place: Hotel Warwick, Philadelphia, Pa., Jan. 9-10, 1958.

Southern—Chairman: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Place: Peabody Hotel, Memphis, Tenn., Jan. 25-26, 1958.

Middle—Chairman: Dr. Francis L. Lederer, 860 N. Lake Drive, Chicago, Ill.
Place: Hotel Harrison, Chicago, Ill., Jan. 13-14, 1958.

Western—Chairman: Dr. Ben R. Dysart, 960 E. Green St., Pasadena 1, Calif.
Place: Beverly-Hilton Hotel, Beverly Hills, Calif., Jan. 18-19, 1958.

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Section Delegate: Gordon Harkness, M.D., Davenport, Iowa.
Alternate Delegate: Dean Lierle, M.D., Iowa City, Iowa.

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President: Dr. Dean M. Lierle, State University of Iowa, Iowa City, Ia.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14, Minn.
Place: Fairmont Hotel, San Francisco, Calif., May 17-18, 1958.
Place: The Homestead, Hot Springs, Va., 1959.

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Secretary: Dr. Robert M. Hansen, 1735 No. Wheeler Ave., Portland, Ore.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, Ill., October, 1958.
Annual Meeting: October, 1958, Chicago, Ill. (Definite time and place to be announced later).

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3Secretary: Dr. Samuel M. Bloom, 123 East 83rd St., New York 28, N. Y.
Meeting: Columbus, Ohio, March 7-8, 1958.

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Annual Meeting: Palmer House, Chicago, Ill.

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Meeting: Nova Scotian Hotel, Halifax, N. S., June 9-11, 1958.

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Secretary:
Meeting: Lima, Peru, 1957.

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Vice-President: Dr. Max M. Kulvin.
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Meeting quarterly (March, May, October and December), on the second Thursday of the month, 6:30 P.M. at Seven Seas Restaurant.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Theodor Hunermann, Dusseldorf, Germany.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadephia 40, Pa., U. S. A.
Meeting: Sixth International Congress of Bronchoesophagology, Philadelphia.

KANSAS CITY SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY.

President: Dr. Clarence H. Steele.
President-Elect: Dr. Dick H. Underwood.
Secretary: Dr. James T. Robison, 4620 J. C. Nichols Parkway, Kansas City, Mo.
Meeting: Third Thursday of November, January, February and April.

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Chairman of Otolaryngology Section: Dr. Howard G. Gottschalk.
Secretary of Otolaryngology Section: Dr. Robert W. Godwin.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P. M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

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Meeting:

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Assistant Secretary-Treasurer: Dr. William F. Murrah, Jr., Exchange Bldg., Memphis, Tenn.
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Meeting:

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Meeting: Palmer House, Chicago, Ill.

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Meeting:

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Jose Gros, Havana, Cuba.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Sixth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Brazil, 1958.

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Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

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Time and place of meeting: October 15, 1957, Palmer House, Chicago, Ill.

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Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Virginia.
Meeting: Roanoke, Virginia, December 6 and 7, 1957.

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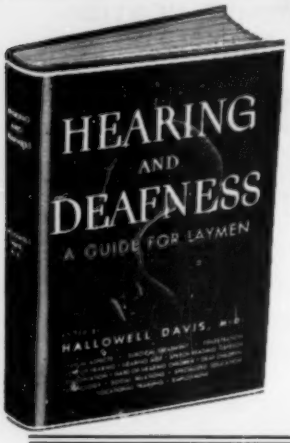

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1. *Riddle, A.C., Jr.: Adrenosem Salicylate: A Systemic Hemostat, Oral Surg., Oral Med., Oral Path. 6:617 (June, 1955).*

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Lewis, H. H.; Frumess, G. M., and Henschel, E. J.: Rocky Mountain M. J. 54:806 (Aug.) 1957.

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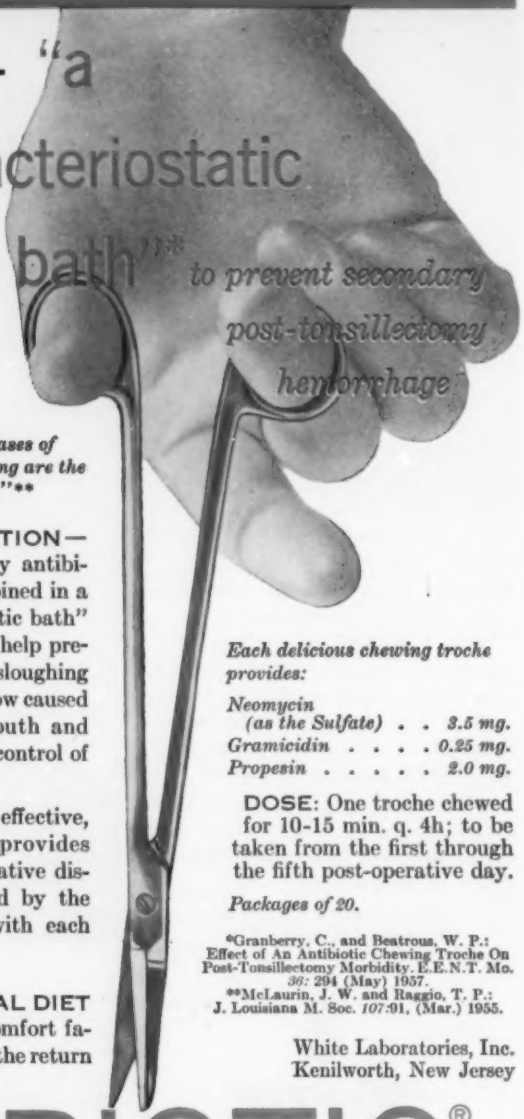

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*Granberry, C., and Beatrous, W. P.: Effect of An Antibiotic Chewing Troche On Post-Tonsillectomy Morbidity. E.E.N.T. Mo. *36:* 294 (May) 1957.
**McLaurin, J. W. and Raggio, T. P.: J. Louisiana M. Soc. *107*:91, (Mar.) 1955.

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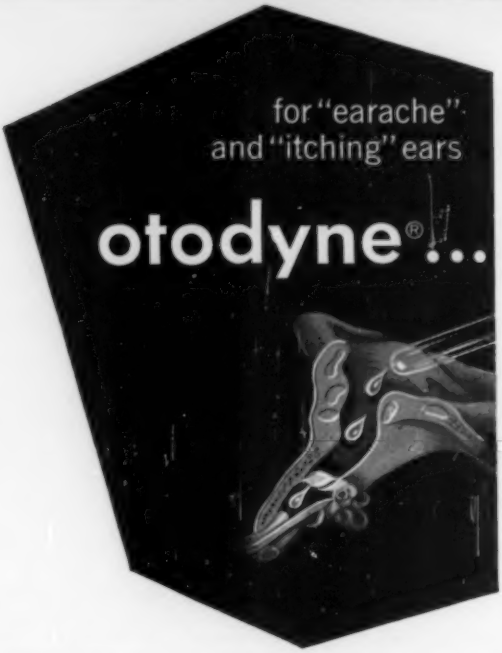
Books Received

Books and monographs received are acknowledged in this column. This notice may be regarded as a return courtesy to the publisher or author. Reviews will be published later as the editors may elect.

Notice of each book or monograph, including title, name of author, publisher, pagination, price, etc., will be presented in these notices so that our readers may have all data at their disposal for further inquiry.

A Synopsis of Otorhinolaryngology. By John F. Simpson, F.R.C.S., Consultant Surgeon, Ear, Nose and Throat Department, St. Mary's Hospital, London, and Paddington General Hospital, etc., Ian G. Robin, F.R.C.S., Consultant Surgeon, Ear, Nose and Throat Department, St. Mary's Hospital, London, and Royal Northern Hospital, London, etc., and J. Chalmers Ballantyne, F.R.C.S., Honorary First Assistant to the Ear, Nose and Throat Department of St. Mary's Hospital, London; Consultant Surgeon, Ear, Nose and Throat Department, Royal Northern Hospital, London; Assistant Director of Audiology Unit, Royal National Throat, Nose and Ear Hospital, London, etc., with a section on Neurology of the Ear, Nose and Throat, by Charles Harold Edwards, M.R.C.P., Consultant Physician to the Neurological Department at St. Mary's Hospital, London; Consultant Neurologist to the Royal National Throat, Nose and Ear Hospital, London. 444 pages with Index and 88 figures. 1957. The William Wilkins Company, Baltimore, Md. Price $8.50.



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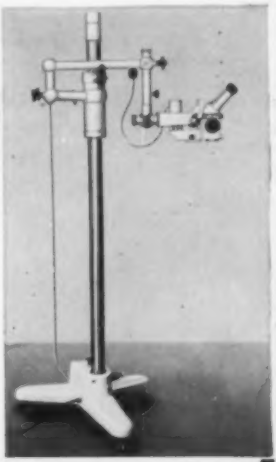

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References: 1. Gutner, L. B., Gould, W. J., and Cracovaner, A. J.: The Effects of Cyclizine Hydrochloride and Chlorcyclizine Hydrochloride Upon Vestibular Function, A.M.A.Arch.Otolaryng. 59:503 (Apr.) 1954. 2. Witzeman, L. A.: Cyclizine Hydrochloride in the Treatment of Vertigo, Eye, Ear, Nose and Throat Monthly 33:298 (May) 1954. 3. Gutner, L. B., Gould, W. J., and Hanley, J. S.: Effect of Meclizine Hydrochloride Upon Vestibular Function, A.M.A.Arch. Otolaryng. 62:497 (Nov.) 1955.

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